THE MEDICAL JOURNAL OF AUSTRALIA

VOL. II.—28TH YEAR. SYDNEY, SATURDAY, SEPTEMBER 20, 1941. No. 12.

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THE TREATMENT OF THYREOTOXICOSIS.[1]

By S. A. SMITH, M.B., Ch.M., F.R.A.C.P.,
Sydney.

IN the present state of knowledge it must be recognized that the ultimate cause of thyreotoxicosis is unknown. Beyond this there are many aspects of the condition that are little understood by the expert. But the general practitioner, although he may be unable to differentiate between various types of the disease, as for instance between disorders of secretion and disorders of colloid storage, or to decide if some forms are due to an increase in thyreoid hormone or to the circulation of an altered hormone, is not in much difficulty usually in recognizing the presence of thyreotoxicosis or the probability of its eventual development. Except in some instances of very early or very mild chronic poisoning and some other conditions associated with ocular signs, the problem of diagnosis is not severe.

The coexistence of several signs or symptoms, among which are palpitation of the heart, tachycardia, tremulousness, increased appetite combined with progressive loss of weight, altered sensitivity to heat and cold, enlargement of the thyreoid gland and the presence of characteristic eye signs, points clearly to the diagnosis, which can be confirmed by the finding of a diminished basal metabolic rate, possibly by certain tests of liver function and by the characteristic response to the administration of iodine.

When once the existence of thyreotoxicosis has been recognized, the choice of methods of treatment is usually not difficult. Since the cause of this condition is not known, our knowledge of treatment is, of course, purely empirical. At the present time we possess no antihormone or antibody to thyreoid hormone for use in combating the disease, although J. H. Means states that such a method of treatment is in the realm of possibility in the future. However, there are two substances which have the power to alter specifically the function of the thyreoid gland, namely, iodine and the thyreotropic hormone of the anterior pituitary. Although knowledge of the thyreotropic hormone is increasing, the stage has not been reached when it may be employed clinically. Therefore, we are limited to one substance, iodine, for specific therapeutic use. All other methods of medical treatment are purely secondary and not specific.

Iodine Therapy.

There are now available the results of a very considerable experience of iodine in toxic goitre. It was in 1820 that it was first used for this disease by Coindet, of Geneva; but Trousseau, in 1863, was the first to recognize beyond doubt the characteristic response to iodine in toxic goitre, his rediscovery being due to a mistake in writing a prescription for iodine instead of digitalis for a patient sufferng from the tachycardia of thyreotoxicosis. He observed the remarkable improvement which ensued, and the return of the tachycardia when he corrected the prescription substituting digitalis. Trousseau's work did not lead to the general use of iodine, and Kocher and his associates in 1910 brought it into disfavour. However, H. S. Plummer in 1923 reintroduced it, and since that time knowledge of its effects has become crystallized and it has now been universally recognized that the response to iodine is characteristic and predictable in almost all instances. Iodine is in fact capable of producing an immediate response in thyreotoxicosis in over 95% of patients.

The phenomena observed after the effective use of iodine in the thyreotoxic patient are:

A. A remarkable improvement in the symptoms, nervous, psychotic and circulatory.

B. A definite fall in the basal metabolic rate in parallel with the symptomatic improvement.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on July 31, 1941.

C. A profound change in the histological characters of the gland, characterized by involution, increase in colloid storage and recession of hyperplasia.

These are the characteristics of the iodine response and the following facts are known:

1. The effect of iodine is on the gland itself, with resulting change in its function and its delivery of hormones to the body.

2. The maximum iodine response is obtained in about ten to fourteen days.

3. It has no lasting effect on the function of the gland, because (*a*) if iodine is discontinued after the full response has been obtained, there is a prompt return to the previous level of thyreotoxicosis; and (*b*) if iodine is continued for a longer period there occurs frequently a return or even an intensification of the thyreotoxic state in spite of the iodine. In fact, iodine has no effect on the progress or duration of the disease.

4. The iodine response may be obtained at any stage of the disease, early or late, and in any form of thyreotoxicosis.

5. Small absolute dosage is all that is required. The iodine needs of the normal body are small, and although it is more essential to normal thyreoid function than to that of other organs, in absolute quantity the requirements of the gland are minute. The minimum dose necessary to produce a maximum response has been calculated as one minim of compound solution of iodine or six milligrammes of iodine per day. It is obvious, therefore, that a dose of nine minims of Lugol's solution per day contains more than the normal iodine content of the gland and is in excess of requirements in the most severe toxicosis.

6. Overdosage beyond the limits of nine or at most fifteen minims of Lugol's solution per day for any length of time does harm. The statement is made by some writers that such overdosage does not matter, since the thyreoid will reject any surplus. This is not the case, because the starved gland seizes iodine with avidity, but its capacity for dealing with it without damage is definitely limited. Overdosage for more than a few days produces that stage of a fibrous brittle organ which is so familiar to the pathologist and so embarrassing to the surgeon, and, most important of all, so disappointing to the patient, who finds the thyreotoxicosis has not disappeared or even been controlled.

In all instances, therefore, small doses of iodine, namely, six or nine minims of Lugol's solution per day, and never more than fifteen, given for a period of fourteen to twenty-one days at the outmost, are of great value when used with discretion. Beyond this the use of iodine becomes misuse.

From these considerations the role of iodine in treatment can be well understood. Although the iodine response is so striking, it is of such short duration that it is useless to demand a prolonged or a curative action. However, it has great value in those circumstances in which a short response is particularly valuable. First among these is the preparation of the patient for operation. The diminution of symptoms, the distinct fall in the basal metabolic rate and the altered anatomical condition of the gland all bring about a state especially favourable to surgery.

Similarly in severe thyreotoxic crises, when the dangers of operation are too great and where the obvious necessity is to tide the patient over a short but critical period, the use of iodine is valuable.

As a definitive form of treatment in itself it has a very limited scope. After its reintroduction in 1923 it was hoped that, combined with rest, iodine might be effective in a large proportion of cases, and it became the practice to use large doses over a relatively long period. Experience, however, proved the error of this, but unfortunately many still persist in using iodine in this manner. It is a common experience to meet patients who have been treated with excessive doses of Lugol's solution for a period of months to their serious detriment. As Rose states:

> Misuse of iodine is the most common and the most unfortunate mistake in the treatment of thyreotoxicosis. Its usual prompt and dramatic effect encourages physicians to continue its use long after its benefits have been wasted, until the patient no longer responds, thus seriously increasing the difficulties of subsequent treatment, since if thyreoidectomy eventually becomes necessary, one of the surgeon's most valuable weapons has been lost and the danger of operation tremendously increased.

This misuse of iodine arises from ignorance of the fact that this substance does not cure the disease or retard its course. It only modifies some of its features, reducing their severity.

Iodine has, however, a small place as a definitive treatment. It is important to recognize that thyreotoxicosis is in very many instances a cyclic disease with exacerbations and remissions. Complete data are not available, but such information as we have indicates that in a certain proportion of patients there is a tendency to spontaneous remission. In some, therefore, suffering from very mild and early toxicity, especially in the colloid goitre of the adolescent, small doses of iodine for a short period may hasten or produce an instant remission and they may be held indefinitely in remission with occasional periods of iodine therapy. Such permanent responses, however, are very rare in patients with well-established toxicity, and it is difficult to identify such cases in advance. The use of iodine in this manner is infrequently indicated and it demands most expert judgement.

Iodine has one further use, namely, in diagnosis. In doubtful instances, if the administration of small doses of iodine with the patient at rest produces a fall in the metabolic rate which rises again on discontinuance of the drug, the presence of thyreotoxicosis is indicated. This is of more value than a simple basal metabolic rate estimation, and the test is on a firmer ground at present than tests of liver function.

Other Forms of Therapy.

The other specific means of treatment are surgical removal of most or whole of the gland and irradiation of the thyreoid and/or the pituitary glands. These I do not propose to discuss. In the absence of any medicinal treatment of a curative nature and in the absence of any antihormone, relief from the symptoms and effects of thyreotoxicosis must be sought by the somewhat crude method of removal of the gland by surgery or its destruction by irradiation. The effect aimed at by each of these methods is the same. In spite of the fact that X radiation entails no immediate risk, it has a limited scope in the treatment of thyreotoxicosis, being generally less effective in the complete control of the toxicity, and it necessitates a much more prolonged period of rest and treatment with the attendant psychological and economic handicaps.

The Effect on the Heart in Thyreotoxicosis.

To the physician a most important aspect of thyreotoxicosis is the effect on the heart. Having regard to the amount of attention which has been given to the clinical characteristics of thyreotoxicosis and of chronic heart disease in the last quarter of a century, it is somewhat remarkable that it is only comparatively recently that the form of mild chronic thyreotoxicosis in which the effect on the heart is the outstanding feature has come to be generally recognized. This lack of recognition was due undoubtedly to the fact that, although the presence of cardio-vascular disease was clear, as auricular fibrillation, paroxysmal or persistent, congestive cardiac failure, alterations of arterial tension or cardiac pain, the evidences of the causal thyreotoxicosis were not obvious, were in fact "masked" or (preferably) "cryptic". It has long been known that "the weak point in any individual's economy may break down under the burden of thyreotoxicosis. In one patient it may be the cardio-vascular apparatus, in another the liver, in a third the psyche". If there is a preexistent coronary sclerosis or diabetes or avitaminosis (*B*), its effects are intensified by the thyreotoxicosis. In such circumstances the removal of the thyreoid poison benefits the patient to a remarkable degree.

Masked thyreotoxicosis is responsible for the precipitation or untimely development of cardiac failure in one form or another in patients of middle age or over, and when it is recognized early and treated effectively results are obtained which cannot be matched in any other form of heart disease.

Diagnosis.

The first step in the diagnosis of this state is to think of the possibility of thyreotoxicosis in every patient who manifests auricular fibrillation, whether the arrhythmia is paroxysmal or permanent, and in every patient in whom cardiac failure has appeared, especially if there is no obvious cause, such as rheumatic carditis or true hypertension. The diagnosis is not reached merely by exclusion, because evidence, positive but slight, of thyreotoxicosis is present in many instances. A raised basal metabolic rate is not always revealed in a single estimation, but is frequently present. A history of loss of weight is usual, tremor and tachycardia, not always constant, may be present, and enlargement of the thyreoid may be so slight as to escape detection. The characteristic reaction to iodine is sometimes shown. It is interesting to note that however long standing the thyreotoxicosis may have been, the thyreoid still responds to iodine just as well as in the early attack in the young patient.

Among many examples there comes to mind a patient, aged fifty years, suspected of carcinoma of the stomach because of loss of weight and attacks of epigastric distress, which under observation proved to be due to paroxysmal auricular fibrillation, and in whom, despite a normal basal metabolic rate and absence of other signs of thyreotoxicosis, total removal of a large nodular retrosternal gland was followed by a complete return to health.

A man of fifty-two years, who had spent the greater part of a year in hospital because of auricular fibrillation and congestive cardiac failure, thought to be due to rheumatic heart disease, but who on routine examination was found to have a basal metabolic rate of +25% and gave a history of loss of weight, was able to resume a life of moderate activity after the total removal of a thyreoid gland which did not appear to be abnormal until histological examination proved the contrary.

A wharf labourer of forty-one years suffered auricular fibrillation after a severe effort at work; no previous signs or symptoms of disease were present except some loss of weight, and he gave no history of acute rheumatism or chorea. He had a blood pressure within normal limits, did not react to the Wassermann test and, after the auricular fibrillation had yielded to treatment, his electrocardiogram was normal, except for a persistent sinus tachycardia.

This man had received compensation for months for "strained heart" until he was sent for examination to a physician whose mind was alive to the possibility of cryptic thyreotoxicosis and who was able by further investigation to establish the diagnosis.

The early recognition of the cardiac damage which may arise from a very mild chronic thyreotoxicosis will lead to the restoration of many patients to a high degree of cardiac efficiency and health after total removal of the diseased gland.

THE SURGEON'S RESPONSIBILITIES IN THYREOTOXICOSIS.[1]

By HUGH R. G. POATE,

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THE prevention of thyreotoxicosis is a question which has exercised the minds of investigators the world over. Except for apparently spontaneous cases of acute Graves's disease, it seems likely that thyreotoxicosis arises in a previously damaged gland.

Prophylaxis.

It is recognized that endemic goitre is prevalent in certain countries or districts, and this applies to Australia just as it does to other parts of the world. In New South Wales we know fairly well the location of these goitre districts, and it seems that the time has come when we should advise the general use of iodized salt and the avoidance of goitrogenic foods. Medical practitioners can play a part in such general measures, but more so in the advice given to patients with adenomatous conditions of the thyreoid, as so many of these develop secondary thyreotoxicosis. The old advice that they need not worry about it unless some trouble develops should be discarded, and in all cases surgical removal is to be recommended. The common age periods for the onset of toxicity in these cases are thirty to thirty-five years and forty-five to fifty years, although many cases occur outside these age periods and even in the seventies.

The great danger in such cases is the insidious onset of thyreotoxicity, which is usually of a low-grade chronic type developing over a period of years without the patient realizing what is happening, until perhaps auricular fibrillation occurs, when he consults a doctor. The lesson to be learned is that early operation is advisable in all cases of adenoma of the thyreoid, whether single or multiple, small or large, and irrespective of age, except perhaps for the later years of life, when other factors require consideration. The recognition and control of hyperthyreoidism as distinct from thyreotoxicosis has been referred to by me in THE MEDICAL JOURNAL OF AUSTRALIA of June 20, 1936, and further experience confirms my views in this connexion; but as time is limited I shall merely refer you to that paper.

Preparation for Operation.

When the diagnosis has been established and the patient placed in hospital, what are our next steps? Preoperative treatment is based upon an exact knowledge of the physiology of the thyreoid gland and of the pathology of its diseases. Measures are instituted at once to control the apprehension and nervous manifestations exhibited by these people. They do not feel ill in the ordinarily accepted sense, and a quiet, confident assurance helps them greatly. Sedatives are necessary, the best being bromides with one of the barbiturates in small doses. As they live at high tension and with all metabolic activities speeded up, there is apt to be a diminution of the alkali reserve of the body, with a consequent tendency to acidæmia. Where there has been any great loss of weight incomplete combustion of fats may result in the formation of acetone bodies. This is aggravated if the blood sugar level falls, as complete combustion of fat occurs only if sugar is being utilized in adequate amounts at the same time. In order to combat any fall in the alkali reserve it is necessary to administer regular and adequate amounts of alkali, of which bicarbonate of soda is the best.

It is an established fact that in the thyreotoxic states the liver glycogen is mobilized and gross depletion of the liver storage may occur. This can be made good only by a liberal diet and the administration of glucose. Quite apart from the restoration of the liver glycogen, we must remember that carbohydrates are antiketogenic and play a part in maintaining the alkali reserve. The end-products of protein digestion are the amino-acids, some of which, of course, pass through the liver to enter the systemic circulation to make good some of the tissue wastage; but the balance undergoes a process of de-amination in the liver, forming urea. From some of the non-nitrogenous residue the liver can form glycogen, and this is of importance for very wasted patients.

From this brief review certain indications as to treatment emerge. Diet is important, and should consist of foodstuffs rich in carbohydrate, rich in protein and poor in fat, of a sufficient caloric value not only to make up for tissue deficiencies, chief of which concerns the liver, but also to give an excess so that the patient gains in weight. It is suggested that the diet should consist of 70% carbohydrate, 25% protein and not more than 5% fat in its caloric composition, and that a minimum of 2,500 to 3,500 Calories per day should be given in preparation for operation.

If any degree of anæmia is obvious or suspected, a blood examination should be carried out, and steps must be instituted to combat the condition, since a poor oxygen-carrying power inhibits the normal physiological reactions and aggravates any existing liver deficiency or tissue damage. These people require a high oxygen supply to

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on July 31, 1941.

their tissues, especially during and after operation, and any anoxia, whether obvious or not, militates against them and slows up their recovery or even lessens their chances.

The Iodine Question.

One of the features in the histological appearance of the thyreoid gland in acute thyreotoxicosis is the absence of colloid and a hyperplasia of the cells in the acini. In the chronic or secondary conditions colloid may be present; but it is locked in the acini by a diffuse fibrosis, chiefly perilobular, and often round-cell infiltration is seen—changes which are indicative of a low-grade inflammatory reaction. This picture means that the iodine needs of the body in general are not being met, and in order to restore colloid formation and supply the combined as well as the free iodine in the blood stream, it is necessary to give iodine by mouth. This must be done carefully, and with due regard to the body needs, since any excess will result in a choking of the thyreoid which seizes avidly on any iodine, just as a starving man is apt to overeat when plentiful supplies of food are put in front of him. No permanent harm will be done by an excess of iodine given for two or three days, but after this an actual thyreoiditis occurs with a great increase of fibrous and round-cell infiltration, not only around the acini but through and around the whole gland. As a result it becomes stony hard and brittle, tissue planes are obliterated, the fibrous capsule adheres to the infrahyoid muscles, and new blood vessels develop in the adventitious tissue, all of which features render operation a bloody, difficult and perilous procedure. Three drops of Lugol's solution three times a day for not more than three weeks is the average dose for optimum results, and it is only in serious cases, in which there is no response to this treatment after seven to ten days, that the dosage may be increased to five drops, but only for a few days.

The Routine Treatment.

In view of these considerations one may now lay down the routine treatment adopted for the average patient in preparation for operation. Our old friend *Mistura Gentianæ Alkalina* with 30 grains of sodium bicarbonate and 10 grains of sodium bromide is given three times a day. One of the barbiturates is given in doses of half to one grain twice a day. Lugol's solution is administered, three drops being given three times a day. Glucose is added to a diet rich in protein, rich in carbohydrate and poor in fat. The patient is encouraged to eat as much as possible, and always has biscuits, fruit, barley sugar *et cetera* at hand. If any anæmia is present, active treatment is necessary to remedy it before the patient can be considered fit for operation. I find it best to explain to patients that the object of this régime is to get them into what may be termed the "safety stage", and once they are there the surgical part of their treatment can be undertaken with the minimal risk.

When is a Patient Fit for Operation?

There is no rule of thumb for the decision as to when a patient is fit for operation. It can be arrived at only after careful consideration of the clinical condition of each individual patient; the response to sedation is evidenced by mental as well as physical rest; the pulse rate must have settled to a stable level; the basal metabolic rate falls usually on parallel lines with the clinical improvement; the kidneys must be functioning normally; the hæmoglobin value must be 80% or over, the liver function satisfactory and the lungs clear. In the majority of cases this happy state of affairs can be achieved in some ten to fourteen days' treatment if the patient is in bed and under strict hospital control.

A consideration of the post-operative fatalities shows that acute liver failure is the chief cause of death; pulmonary complications, such as atelectasis, pulmonary œdema or pneumonitis, claim a percentage of deaths, while other causes are uncommon.

Great care must be exercised with those patients who do not respond to routine treatment, as it is always among these that the fatalities occur. Rarely one meets with a fulminating type of acute thyreotoxicosis which seems predestined to a fatal issue within a few months, despite any form of treatment. I should estimate its frequency as about 1 in 1,500 cases, and have seen it more often in the male. It is generally associated with an enlarged thymus gland, and in the liver extensive degenerative changes and often areas of focal necrosis are found. More often one sees the sthenic young person, who has a persistently high basal metabolic rate with a very large hyperplastic thyreoid and an irregular pulse rate, from 100 to 130 per minute or more. Such patients are best treated by a full course of deep X-ray therapy—say ten daily treatments of about 200 r, the actual dose depending on the condition of the patient. The administration of sedatives and iodine should be continued during this treatment, but the iodine should be omitted for two months after it. As a rule they then show considerable clinical improvement and can be subjected to operation after a further course of the routine treatment. If their condition does not satisfactorily settle down, a further course of deep X-ray therapy is indicated.

When elderly patients are resistant to treatment, one generally finds a multiple adenomatous condition present, which is not nearly so responsive to deep X-ray treatment as is the hyperplastic thyreoid gland; if their condition is not satisfactory after a second course of this treatment, it is wiser to withhold any operative measures.

Auricular fibrillation in itself is no bar to operation—in fact, it is a direct indication for surgical treatment, as in over 80% of cases normal rhythm is restored within two days to two months, and the operative risk in this class of case is only 0·5%.

Acute Liver Failure.

Acute liver failure is a condition which is directly consequential to operative intervention in certain cases. It is characterized by a steadily rising pulse rate and temperature, associated with early restlessness, then stupor going on to coma. The blood urea level rises, renal secretion fails and death ensues in twelve to thirty-six hours, the temperature being 106° to 108° F.; the usual picture of peripheral circulatory failure is present. It is a tragic condition, and seems to defy all forms of treatment in the majority of cases. Predisposing causes are prolonged ill health, alcoholism, anæmia, wasting, impoverished diet and prolonged iodine medication. It is especially apt to occur in a liver already damaged by either cellular degeneration or fatty infiltration, and is most commonly met with after operations on the biliary tract or for thyreotoxicosis; but it is occasionally seen after such simple procedures as interval appendicectomy, herniorraphy, oophorectomy *et cetera*. One may say that in most cases it is a preventible condition, as except in emergency cases it can occur only if the patient has been improperly prepared for operation and there has been neglect of or error in the evalution of the liver function.

Liver Function.

Of the many functions of the liver, the two which most concern us in dealing with the thyreotoxic patient are the glycogenic and the detoxicating functions. It is seldom that we are concerned with the excretory function, unless there is an associated toxic hepatitis; thus tests depending on the excretion of the bile, such as the Van den Bergh reaction, the icterus index and the serum bilirubin phosphatase, cholesterol or cholesterol esters do not concern us. The deaminating function is generally the last to be involved to any appreciable extent, so that the estimation of the amino-acids in the blood is of little or no value in thyreotoxicosis.

It seems now to be generally accepted that the hippuric acid excretion test as a guide to the detoxicating function of the liver has proved itself the most reliable. It has the added virtue of being cheap and easy to carry out, and the only conditions in which it may be misleading are in renal disease or in states of dehydration, for example, vomiting, diarrhœa or low fluid intake.

Of all tests of glycogenic function the blood galactose tolerance test seems to be the best, and in thyreotoxicosis it is of greater diagnostic import than even the estimation of the basal metabolic rate; but it is rather expensive and requires a considerable amount of apparatus and the services of a skilled biochemist.

The utilization of galactose has no connexion with the secretion of bile and depends entirely on the liver, as the muscles cannot take it up as they do lævulose. Its greatest value in thyreotoxicosis is the demonstration of possible hepatic damage in a disease in which its presence is uncertain or difficult of confirmation by other measures. As an illustration of its value in differential diagnosis I may mention the following cases.

A woman, with many of the symptoms of chronic thyreotoxicosis, had a basal metabolic rate of +26%. The curve produced by the galactose tolerance test was below normal. She was found to be suffering from chronic myeloid leuchæmia.

Another young woman with many symptoms of chronic thyreotoxicosis had a disorder which was difficult to determine clinically from sympatheticotonia, except for a basal metabolic rate of −11%. The curve produced by the galactose tolerance test was below normal, and this settled the diagnosis by confirming the basal metabolic rate.

A girl suffering from diabetes, an enlarged thyreoid and general symptoms of mild thyreotoxicosis, had a basal metabolic rate of +15%. The curve produced by the galactose tolerance test was below normal, and this showed that her symptoms were part of her diabetic syndrome.

An elderly man with auricular fibrillation had a goitre of many years' standing; but thyreotoxicosis had not been suspected. His basal metabolic rate was +35% and the galactose tolerance test produced a very high curve. He died shortly afterwards from cardiac failure.

The greatest value of the test as a diagnostic method will be found in those states of nervous apprehension and sympathetic imbalance which so closely mimic thyreotoxicosis, and in those cases associated with auricular fibrillation or other symptoms and signs of thyreotoxicosis in which the thyreoid gland is not palpable.

Pre-Operative Danger Signs.

Chief among the pre-operative danger signs is the failure of patients to settle down after two weeks of the routine treatment. They remain jerky in their movements, tremulous, restless, apprehensive and hyperpnœic. The pulse is irregular in rate, jumping without reason from 80 or 90 to 110 or 120 per minute. Generally the thyreoid gland is of the large hyperplastic type and does not become firm with iodine therapy. The basal metabolic rate may fall at first, but does not remain on a stable level. The galactose tolerance test usually produces a high curve; but the result of the hippuric acid excretion test may be 80% or over and usually shows improvement up to 100%, so that this in itself is no guide as a safety factor in these cases. The very dangerous types of case are those associated with an initial result of the hippuric acid excretion test below 50%; this may gradually improve up to 80% or over and may lead to the feeling that the patient is safe; but it is not wise to undertake operation until the result of this test is over 80% for at least two weeks. I consider it unwise to operate when the result is below 70%, no matter how well the patient may seem, as in most cases it is only the matter of a couple of weeks before the result will be up to the full normal. Patients who have had a recent loss of weight of any considerable extent, especially if anæmia is present, and those of the frail elderly type with large thyreoids require special care in the evaluation of the operative risk.

The Anæsthetic Agent.

After his summing up of the patient's condition the surgeon should discuss the choice of anæsthetic agent with a competent anæsthetist. Of late years I have practically given up local analgesia in favour of nitrous oxide anæsthesia.

Adequate premedication is essential, and for routine work I believe that paraldehyde in adequate dosage is the safest; I certainly prefer it to "Avertin" if there has been any evidence of liver damage. I do not recommend the use of any of the barbiturates, as thyreotoxic patients do not stand large doses at all well. "Omnopon" or morphine may also be required; but do not use atropine, as it accentuates the sympathetic imbalance always present. The great essential is a proper appreciation of the oxygen requirements of these patients, and so long as a high percentage of oxygen is maintained it seems immaterial what gas is used or whether some ether is given.

The Operation.

As I have in preparation a series of illustrations depicting the details of my own technique, I shall not deal with this now; but I wish to emphasize the need for gentle handling of the tissues, for painstaking control of hæmorrhage, for expert assistants and for celerity of action. I have found no virtue in preliminary ligation of the superior thyreoid arteries, and I feel that in cases in which it has been performed the patient's condition could have been improved by the means already outlined, and in any case such patients are obviously not safe operation "risks". Moreover, its value is hard to estimate anatomically, for the inferior thyreoid artery is the functional artery of supply to the gland tissue.

If there is any difficulty in removing the right lobe of the gland or any undue hæmorrhage, or if the surgeon is at all uneasy as to the patient's not being a "good risk", it is wise to stop and complete the operation at a second stage some ten to fourteen days later.

Post-Operative Treatment.

Your nurses must be carefully instructed in maintaining an efficient airway and in the proper administration of oxygen, as anoxia to any considerable extent may mean death. Do not use "Carbogen", as it is the oxygen which may be needed, and in the highest percentage possible. The usual tents are useless, and if the "B.L.B." mask is not available, the best way to give the oxygen is to slip a catheter, not less than six millimetres in diameter, through the nostril as far back as the naso-pharynx, fix it with adhesive strapping to forehead or cheek, and then connect it with the oxygen supply; this ought to have a flowmeter, so that five litres per minute can be given for whatever time is desired. This technique and rate of flow ensure that the patient receives about 50% oxygen, which doubles the normal alveolar content and is of value. This adequate supply of oxygen may be required at varying intervals for the first twenty-four hours or longer if the patient shows any signs of impending liver failure or if any pulmonary complications develop. Do not wait for cyanosis to develop, as it is a very late indication of anoxia; but remember that hæmorrhage, shock and the increased metabolic rate following operation all lead to an oxygen want in the tissues.

Next in importance to oxygen is the necessity for implementing and maintaining the fluid balance. As a routine measure the patients are given six ounces of glucose (10%) and bicarbonate of soda (1%) in normal saline solution on their return to bed and at intervals of four to six hours until such time as they will take an adequate amount of fluids by mouth. If the pulse rate and temperature show any undue tendency to rise, an intravenous drip administration of 10% dextrose solution should be given.

Restlessness must be controlled by injections of plain morphine, at intervals sufficient to secure rest. As soon as the patients will take them, glucose drinks are given by mouth, with as much nourishing food as is possible, so as to combat the drain made on the body reserve of glycogen during the stage of post-operative reaction. Alkali and bromide are also given as early as possible, and their administration is maintained until the reactionary stage is over—that is, for five to seven days.

In the case of elderly patients or of those in whom it has already been obvious, a watch must be maintained for fibrillation, and at the first sign, or sometimes before they leave the operating theatre, an intravenous injection of 0·5 milligramme of "Digoxin" is given and repeated as may be necessary. If fibrillation persists after thirty-six

hours, this drug may be given orally in doses of 0·25 milligramme every four hours until a regular rhythm returns. I seldom find it necessary to use quinidine, except when "Digoxin" fails after ten or twelve days, and then use it only after consultation with a physician, for it is a very powerful drug.

If the patient has been properly prepared there should be no need to give iodine after operation, and in any case I cannot understand what value there can be in giving drachm doses of Lugol's solution *per rectum*. If a subtotal thyreoidectomy has been performed, iodine will "choke" the remnant of gland left; and if total thyreoidectomy has been performed there is no gland to utilize it.

To save any scarring of the skin stitches are removed on the fourth day and a light dressing is then applied. Patients may be allowed out of bed after the pulse rate has been stabilized and normal for four or five days, and may leave hospital some four days later, on the understanding that they must rest in peace and quiet for two or three weeks and report back for a check examination about six to eight weeks after operation. This allows one to advise them as to whether any further period of medical treatment is required.

Conclusion.

As it seems that surgery gives the quickest and best results in the treatment of thyreotoxicosis, the earlier such patients can be referred for operation, the safer it can be made for them and the easier it is for the surgeon.

Those who propose to undertake the surgery of this condition must make themselves *au fait* not only with the anatomy of the thyreoid gland, but also with its physiology and with the various pathological conditions which may be associated with the disease.

Only by careful preparation of the patient, by evaluation of the operative risk and by an alert post-operative supervision can the mortality rate be kept down to a reasonable level.

RADIOTHERAPY IN THYREOTOXICOSIS.[1]

By CHARLES DE MONCHAUX,
Sydney.

THE rational treatment of toxic goitre, or thyreotoxicosis, as it is now called, depends, of course, on its essential causation and on the resultant basic pathology of the disease. So the question at once presents itself: Is thyreotoxicosis essentially an intrinsic condition of the thyreoid gland, or is it primarily due to some cause or influence external to the gland, such as, for example, some dysfunction of the pituitary gland or some profound disturbance of the autonomic nervous system? On the answer to this question will depend the rational, or scientific, treatment of thyreotoxicosis. Whatever be the *causa causans* of toxic goitre, however, the noteworthy success achieved by our present methods of treatment, whether medical, surgical or radiotherapeutic, all of which are designed to attack the thyreoid gland directly, strongly suggests that some factor or factors intrinsic in the thyreoid itself have much to do with the ætiology of this fairly common disease. In all forms of goitre, or thyreoidal hyperplasia, whether associated with thyreotoxicosis or not, the thyreoid gland, as the factory, storehouse and purveyor of thyroxin (an essential commodity in the bodily economy), would seem to be the "common denominator" and the all-important consideration. And just as the gravity of the toxic forms of goitre is measured clinically by the effects and the degree of thyreoidal toxicity, so the degree or intensity of thyreotoxicosis becomes the chief guide and criterion in the three different methods of treatment.

Apart from the simple forms of thyreoiditis and the malignant varieties of thyreoidal neoplasia, which may, of course, give rise to enlargement of the gland, our present interest and concern are with those forms of true thyreoidal hyperplasia, which cause more or less enlargement of the thyreoid gland, which may or may not be associated with thyreotoxicosis, and which are included commonly under the generic term of goitre. According to the opinion most recently expressed by pathologists, which appears to have the sound bases of histology and physiology, three main forms or varieties of goitre, or thyreoidal hyperplasia, emerge, and these are made the basis of a simple classification, which will guide us in the matter of logical treatment, whether by purely medical, surgical or radiotherapeutic means. This broad classification of goitre is consistent with the most recent opinion, as expressed in the latest edition of Boyd's "Pathology of Internal Diseases".[(1)] Here is the classification suggested:

1. Diffuse non-toxic goitre, in which histologically there is more evidence of involution than of hyperplasia, as a rule. This variety is non-toxic and may be said to be physiological rather than pathological. There are two main varieties of this: (i) parenchymatous and (ii) colloid.

2. Discrete nodular (or lobular) goitre, or the so-called "adenomatous" goitre—a misnomer, for the lesions are not true adenomata. There are two varieties of this form: (i) the non-toxic, with involution predominant, and (ii) the toxic variety, with hyperplasia predominant.

3. Diffuse toxic goitre, with evidence of more hyperplasia than involution histologically. This form is toxic and pathological: the so-called exophthalmic goitre, Graves's disease, Basedow's disease *et cetera*.

As thyreotoxicosis is the broad indication for treatment by irradiation, it will be seen at once that the diffuse forms of non-toxic goitre and the non-toxic variety of nodular goitre are not the concern of the radiotherapist, their treatment being entirely medical and/or surgical. Radiotherapy has, however, an important role in the treatment of the two toxic varieties, namely, diffuse toxic goitre or Graves's disease, and the toxic form of discrete nodular goitre. These two together represent and constitute what is known as thyreotoxicosis. The trend of modern thought among pathologists would seem to be towards the convergence, rather than divergence, of these three fundamental forms of thyreoidal hyperplasia. It is suggested that these three varieties are not so definite and clear-cut as they might first appear. One variety may emerge or develop into another, and in all three varieties there may commonly be found, on structural or histological examination, all grades and proportions of hyperplasia and its "negative phase", called involution. So that the crux of the matter, in all three forms of goitre, is not so much the abnormal structure of the gland as its impaired function, which may mean functional underactivity or overactivity, the latter abnormal state of the gland giving rise directly to the condition known as thyreotoxicosis. So it would seem possible to reconcile and correlate the various forms of goitre, and especially to bring into line the two toxic varieties, the discrete or nodular (so-called "toxic adenoma") and the diffuse or exophthalmic.

Apropos of the question of terminology in thyreoidal hyperplasia, I should like, in parentheses, to enter a protest against the continued use of that clumsy and imprecise term, "hyperthyreoidism". Though employed usually to express the idea of functional overactivity of the thyreoid, this vague term might just as well mean simply structural overgrowth, or thyreoidal hyperplasia (not necessarily associated with thyreotoxicosis). And since we have the good word "goitre", sanctioned by age and long usage, to express the idea of structural overgrowth or hyperplasia, and the new but precise term "thyreotoxicosis" to represent functional overactivity or "hyperfunction", as it is sometimes miscalled, I would deprecate strongly the use of the loose term "hyperthyreoidism", which implies either structural or functional excess, and which sounds more like some new, ephemeral religion than anything else—the cult of the thyreoid, as it were, or, perhaps, an undue and excessive devotion to the shield-like qualities of the thyreoid gland! (Greek, *θύρεός*, a shield).

Before discussing the more particular indications for radiotherapy in toxic goitre, discrete or diffuse, I wish to

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on July 31, 1941.

say a few words about the biological action and effects of irradiation in this disease. Normal thyreoid tissue is more radioresistant than that of the thyreoid gland exhibiting more or less hyperplasia together with thyreotoxicosis. The comparative radioresistance of the normal thyreoid gland may be adduced from the absence of any noticeable degree of hypothyreoidal activity following the treatment by radiotherapeutic methods of lesions in the neck, in close proximity to the thyreoid gland. Heavy doses of X rays (or γ rays from radium) may be given, for example, in the treatment of carcinomata of the larynx or hypopharynx, and though the thyreoid gland must receive a fair amount of irradiation in such cases (at least from secondary or scattered radiation, if not from unavoidable primary irradiation), one never or very rarely with the use of modern techniques sees any subsequent signs of myxœdema or even any mild untoward hypothyreoidal effects. So we say that the normal thyreoid gland is relatively radioresistant. The tissues of the thyreoid, however, in the abnormal state of thyreotoxicosis are much more radiosensitive, and this increased sensitivity or vulnerability refers, of course, to the cellular tissue of the gland, which in the toxic forms of goitre shows definite evidence of hyperplasia. This difference in radiosensitivity suggests at once that the actual cells in the hyperplasia of toxic goitre are much more embryonic and less differentiated than those lining the acini in the normal thyreoid gland. This view, incidentally, harmonizes completely with our present notions of tissue cells and radiosensitivity, and with the usually increased radiosensitivity of the cells in practically all forms of epithelial hyperplasia.

Mild to moderate doses of X-ray energy, given for short to moderate periods, are the rule in the treatment of toxic goitre by radiotherapy. There is no need at all to give large, destructive doses of radiant energy, such as one would administer, for example, to a carcinoma of the thyreoid. Briefly expressed, the biological action is, then, one of inhibition rather than destruction. The amount of hyperplasia is reduced and so there is a corresponding diminution in the functional activity of the gland. The acinar hyperplasia is lessened, the secretion of "colloid" is decreased (and so the production and supply of thyroxin), and the degree or intensity of thyreotoxicosis is diminished, with corresponding amelioration of the patient's condition.

Indications for Radiotherapy.

The specific indications for radiotherapy in thyreotoxicosis may be divided into primary and secondary, and are as follows:

I. Irradiation is primarily the indication in the following circumstances:

(*a*) In thyreotoxicosis in children and young people, especially when associated with much nervousness.

(*b*) In thyreotoxicosis (usually mild) occurring in old people. Both these conditions are (or should be) contraindications to surgical treatment.

(*c*) In inoperable cases. This group includes all cases in which, for any reason whatsoever, apart from age, a surgical operation is deemed to be inadvisable or undesirable.

(*d*) In the treatment of patients who refuse a surgical operation.

(*e*) In anomalous or "border-line" cases, and especially those rather vague cases in which psychic elements or psychotic features predominate. In this group (*e*) very mild doses of irradiation may be given, often rather as a psychotherapeutic measure than for any real physical or biological effect on the gland itself.

II. Irradiation is indicated secondarily:

(*a*) As a pre-operative measure, in many cases, to reduce toxicity and improve the surgical chances.

(*b*) As a post-operative measure, in all cases of post-operative recurrence.

In Group I radiotherapy is the primary, and should be the sole, treatment; in Group II it is combined with surgery, as an auxiliary form of treatment, either before or after operation. Irradiation is contraindicated in all cases of crisis and extreme toxicity (acute thyreotoxicosis) and also whenever goitre is associated with pressure symptoms. With regard to the pre-operative treatment of toxic goitre by X rays (Group II (*a*)), one must say something about the alleged fibrotic or sclerotic effect of irradiation on the thyreoid gland. With the modern "divided-dose" technique, this "effect" is a myth or an illusion. It simply does not happen when X-ray treatment is given according to modern principles and technique. Also the alleged surgical impediment of a hyperæmic "operation field" can easily be avoided if a sufficient interval (let us say, at least two months) is allowed to elapse between the completion of irradiation and the operation.

Dosage, Technique and Factors Used in X-Ray Treatment of Toxic Goitre.

As I have already stated, mild to moderate doses are given, and this applies both to the individual doses and to the total dose administered in a course of treatment. The total dose need never, or very rarely, exceed 1,500 *r*, and 1,800 *r* represents, I think, an outside limit for the total dose. I give, as a rule, a total of 1,200 *r* to 1,500 *r*, divided into eight to ten doses of 150 *r* each, not more than three such doses being given weekly. That is, I favour an interval of at least one day between individual treatments and so the course lasts about three weeks, as a rule. I seldom give more than 150 *r* at a session, 200 *r* being the limit for an individual dose. Two anterior fields are commonly used, the treatments being given alternately to the left and right lateral thyreoid regions (that is, over the left and right thyreoidal lobes); in this way half the total dose is given to each side. All doses are measured on the skin in *r* units (the *r* unit is the fundamental unit of quantity of X-ray energy). At least two months should elapse between courses of X-ray treatment. I repeat that there is never any need to give heavy doses of X-radiation in the treatment of thyreotoxicosis.

The treatment factors used (by me, at least) are as follow: 40 to 50 centimetres (usually 50 centimetres) skin-focal distance; 10 to 15 milliamperes; 180 to 190 kilovolts (no great penetration or super-voltage is necessary to reach any or every part of the thyreoid gland); and a filtration of 0·5 to 0·75 millimetre of copper *plus* one millimetre of aluminium. It is preferable sometimes to have a thyreotoxic patient resting in bed during the course of radiotherapy, but this is not always necessary. Some patients go through with their treatments quite well as ambulant or out-patients, and here again the simple criterion or determining factor is the degree or intensity of thyreotoxicosis.

With moderate dosage and with modern technique, there is no danger of severe skin reactions or of untoward skin sequelæ, and there is no risk of myxœdema or of tetany (the parathyreoids being, of course, irradiated, to some extent, along with the thyreoid). Occasionally, in the presence of a substernal extension of the thyreoid gland (confirmed by X-ray examination), it may be necessary to employ a third field of irradiation, directing the rays to the substernal extension through a central manubrial port. Because some authorities incriminate the thyreotropic hormone of the pituitary as a possible factor in the ætiology of thyreotoxicosis, some radiotherapists recommend and employ irradiation of the pituitary gland; but I have had no experience with this procedure, believing that the possible harmful effects to the function of this important "conductor of the endocrine orchestra" do not justify its irradiation, on purely hypothetical grounds, in the treatment of toxic goitre. Most radiotherapists deprecate the administration of iodine *pari passu* with X-ray therapy. Apart from any other reason, the giving of iodine during a course of irradiation of the thyreoid militates against a true assessment of the relative value of these two therapeutic agents, each of which has much the same rationale, namely, the induction of involution and the reduction of glandular overactivity.

The Response to and Results of Radiotherapy in Toxic Goitre.

Considerable improvement is often observed after one course of X-ray treatment (this is, of course, the rationale

of pre-operative irradiation), and in many cases a second or even a third course of X-radiation is justified and indicated. A small minority of patients do not respond well to radiotherapy. Pfahler, one of the doyens of American radiotherapy, has had a long experience (more than thirty-five years) in the treatment of thyreotoxicosis by irradiation, and so I shall conclude by quoting some of his opinions and results, as expressed in a fairly recent article on this subject, published in the journal *Radiology* of January, 1940.[2] Pfahler believes that irradiation is indicated in all cases of thyreotoxicosis in which the patient is not in crisis or is not suffering from definite pressure symptoms. He states that thousands of patients with thyreotoxicosis have now been treated (by radiotherapy) and recorded, by various authors, including himself, and that the results in general have shown about 65% of cures, about 20% to 25% of patients with marked improvement, and about 10% of failures. That is, about 90% of the patients treated have been restored to a state of what he calls "working ability". This, he goes on to state, is approximately the result obtained by surgical methods of treatment. Pfahler then enumerates the following advantages of irradiation treatment:

1. The fear of operation is eliminated and, therefore, the patient is more likely to come under treatment early and before cardiac damage has taken place.
2. If patients are treated reasonably early, they are not interrupted in their occupation. (That is, there is no need for them to be in hospital.)
3. There is no pain or shock, and no great inconvenience, if the condition is treated reasonably early.
4. Patients with advanced disease or serious heart complications may be treated without shock.
5. There is no risk of mortality from X-ray treatment.
6. There is absence of scars or keloid formation.
7. The end-results are approximately equal to those obtained by surgery.

Exacerbation of symptoms after irradiation has occurred rarely in Pfahler's cases and, in his experience, this has been insignificant and no more than might occur from time to time, quite independently of treatment. Myxœdema has not occurred in any of his cases. Slowness of response to irradiation has been raised as an objection, but, as Pfahler points out, one usually sees some improvement at the end of a month and more marked improvement at the end of two. If the patient is treated reasonably early, a slower recovery is not a serious objection; it may even be an advantage. Pfahler is convinced that it is not advisable to use iodine in conjunction with X-ray therapy; he prefers indeed to treat a patient who has had no previous iodine treatment. He states finally that irradiation is probably of value in preventing the development of thyreoidal carcinoma, especially in the discrete form of toxic goitre (so-called "toxic adenoma").

References.

(1) W. Boyd: "Pathology of Internal Diseases", 1940.
(2) G. E. Pfahler: "Roentgen Ray Treatment of Hyperthyroidism, *Radiology*, January, 1940.

THE TECHNIQUE OF SUBFASCIAL THYREOIDECTOMY.

By HUGH R. G. POATE,

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IN order to understand the basis of the operative technique now presented, it is necessary to have a clear concept of the anatomy of the cervical region in general and of the deep cervical fascia in particular. I have given a detailed description of it in *The Australian and New Zealand Journal of Surgery*, Volume X, July, 1940, at page 48, to which reference should be made for any points of special interest, and in this communication I shall mention only those features which will give a clearer understanding of the operation sketches which have been so ably delineated by Mr. Farrell.

The Deep Cervical Fascia.

The usual text-book descriptions of the deep cervical fascia are somewhat confusing to the surgeon who depends upon fascial planes in his operative work, and it was not until Meyers and Macpherson, of Brisbane, published their work on this subject in THE MEDICAL JOURNAL OF AUSTRALIA in 1938 that a description was given of the cervical fascial

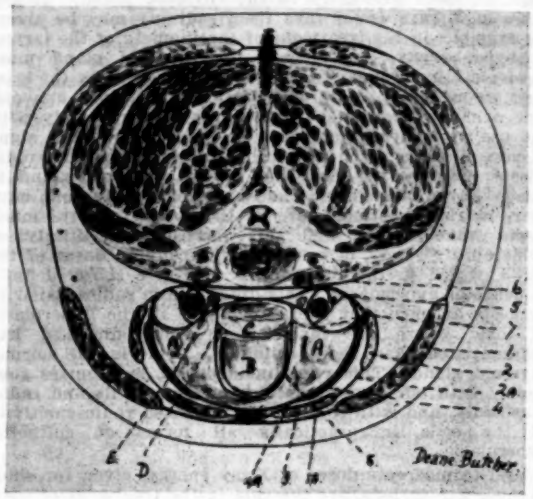

DIAGRAM I.—Diagram of the deep fascia of the neck. (After Meyers and Macpherson.) 1 = sterno-mastoid muscle; 2 = omo-hyoid muscle; 3 = sterno-hyoid muscle; 4 = sterno-thyreoid muscle; 5 = carotid sheath; 6 = paravertebral fascia; 7 = œsophageal fascia; 8 = tracheal fascia; A = thyreoid gland; B = trachea; C = œsophagus; D = inferior laryngeal nerve; E = inferior parathyreoid gland; 1A = sterno-mastoid fascia; 2A = sterno-omo-hyoid fascia; 4A = sterno-thyreoid fascia.

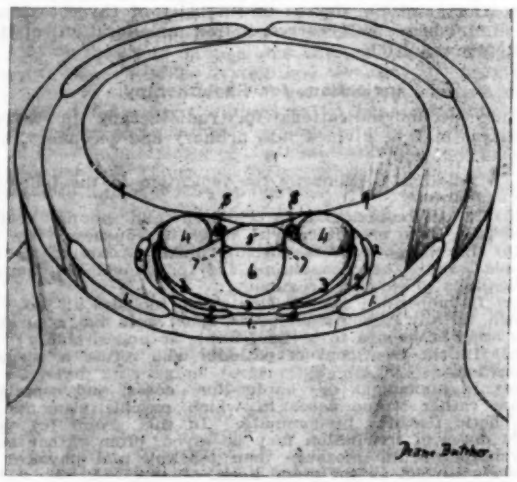

DIAGRAM II.—Schematic representation of the fascial layers of the visceral compartment of the neck. 1 = sterno-mastoid fascia; 2 = sterno-omo-hyoid fascia; 3 = sterno-thyreoid fascia; 4 = carotid sheath; 5 = œsophageal fascia; 6 = tracheal fascia; 7 = space for inferior laryngeal nerve; 8 = inferior parathyreoid gland; 9 = prevertebral fascia.

planes which was in accord with the findings of the surgeon. The fundamental concept of their description lies in the fact that every muscle is enveloped in a fascial envelope, which is attached to bone or cartilage

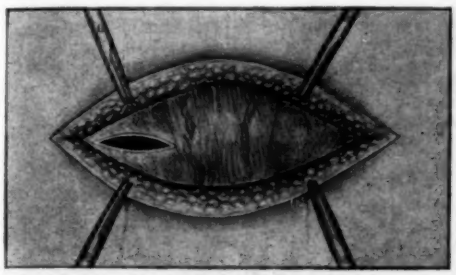

FIGURE I.—The first assistant stands opposite the surgeon, and the second assistant between the surgeon and the head of the patient. The skin and subcutaneous tissue are divided down to the sterno-mastoid layer of fascia. This is incised over the right sterno-mastoid muscle and as far as its anterior border. All small arteries or other bleeding points are caught in artery forceps.

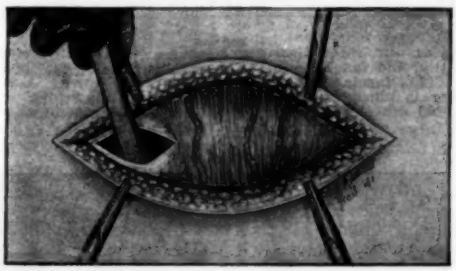

FIGURE II.—With the handle of the scalpel the sterno-mastoid fascia is pushed up and down so that the muscle fibres are cleanly exposed.

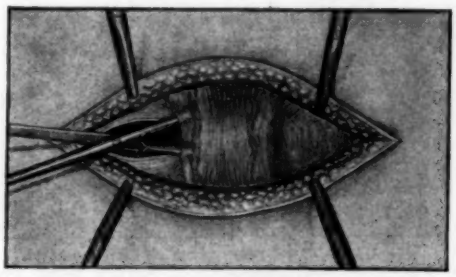

FIGURE III.—A pair of artery forceps is then worked through to or under the sterno-omo-hyoid fascia beneath the anterior jugular veins.

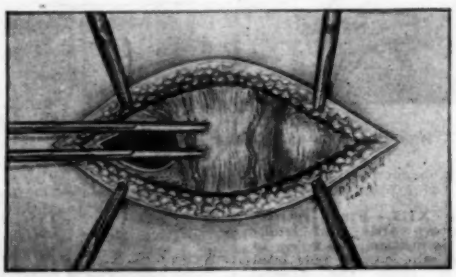

FIGURE IV.—The veins are then clamped, divided between forceps and tied off. If there are large veins or if much fat is present, it is advisable to divide the sterno-mastoid layer of deep fascia over them and the veins can then be seen clearly before they are clamped and divided.

only at the origin and insertion of the muscle, or to the fascial sheaths of great vessels where muscles cross or are in close contact with such structures. These fascial envelopes must therefore correspond with morphological planes—a fact of importance when one considers the relationship of the thyreoid gland, the external and inferior

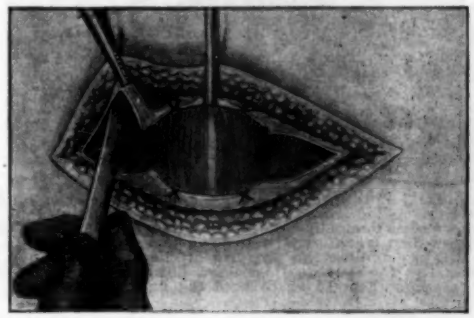

FIGURE V.—When the veins have been clamped and divided, the sterno-omo-hyoid fascia has been incised and the left sterno-mastoid muscle has been exposed in the same manner as in Figures I and II, its fascia is freed upwards from the muscle for several centimetres by pushing the handle of the scalpel under it. The deep fascia is caught in the mid-line by an Allis forceps, which is held by the second assistant in the left hand.

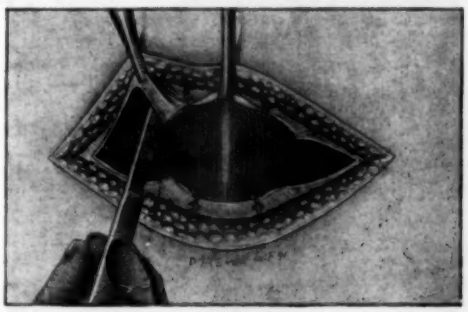

FIGURE VI.—The sterno-mastoid fascia is then lifted up with dissecting forceps and divided along the anterior border of the muscle, first on the right side and then on the left. Then the whole flap is lifted up and cleared from the infrahyoid muscles, the Allis forceps being moved to its deep surface at the point where the fascia has been stripped from the sterno-hyoids.

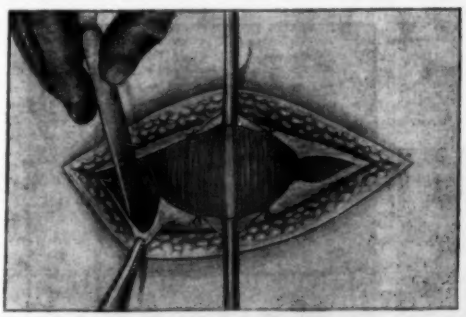

FIGURE VII.—The lower flap is now developed. The deep fascia is caught by Allis forceps in the mid-line and held taut by the first assistant in his left hand. The sterno-mastoid fascia is freed from the muscle by pushing the handle of the scalpel downwards.

laryngeal nerves and the parathyreoid glands to the fascial planes in the lower cervical region.

I shall consider only the region of access to the thyreoid gland. Beneath the skin and subcutaneous fatty tissue in which the platysma muscle and small blood vessels are

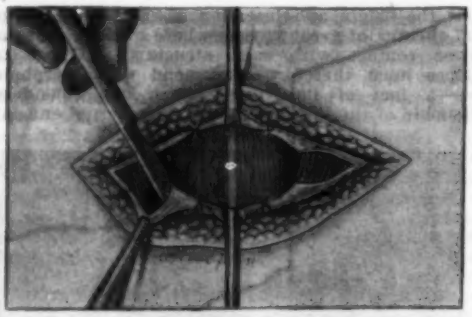

FIGURE VIII.—The fascia is divided along the anterior border of the sterno-mastoid muscles and the infra-hyoid muscles are freed well down so as to mobilize the lower flap for retraction. The Allis forceps is then transferred well down under the flap and a firm hold is taken against the pressure of the first assistant's index finger. The forceps are then fixed down to the sheet on the patient's chest so as to hold the flap well retracted.

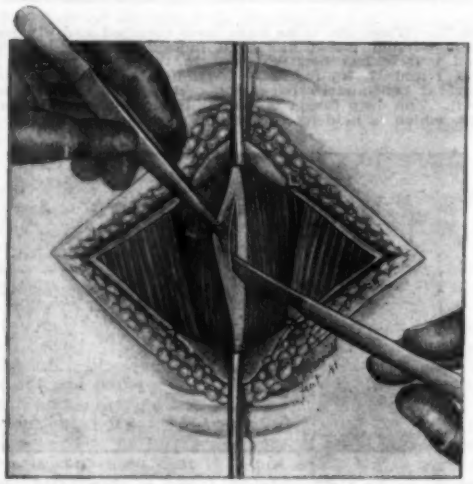

FIGURE IX.—The deep layer of the sterno-hyoid fascia is divided in the mid-line for the full length of the exposed area, and the muscle is lifted up so that the anterior border of the sterno-thyreoid muscle can be seen shining through its covering fascia.

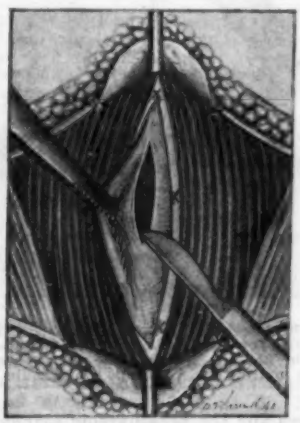

FIGURE X.—The anterior border of the sterno-thyreoid muscle is seized and lifted up with the dissecting forceps and its fascia is opened along the edge of the muscle or just under it, the thyreoid gland thus being exposed about 1·0 centimetre from the mid-line. (Note cut edge of sterno-hyoid fascia, x x x.)

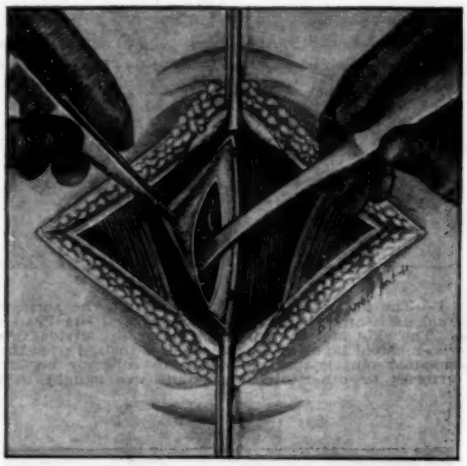

FIGURE XI.—A blunt dissector is then used to run beneath the sterno-thyreoid fascia over the lateral surface of the lobe of the gland. This fascia is then well freed up and down. A Kocher's retractor is placed so as to retract this fascia along with the infrahyoid muscles, and is held in the right hand of the second assistant.

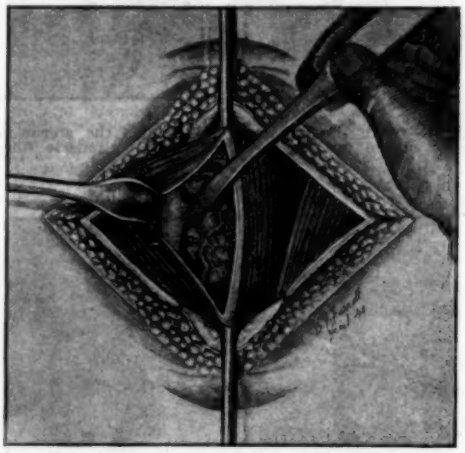

FIGURE XII.—The blunt dissector is worked down over the lateral aspect of the thyreoid gland towards the upper pole. Care must be taken to see that the deep part of the sterno-thyreoid muscle is cleared with its fascia from the gland, which is then separated from the carotid sheath.

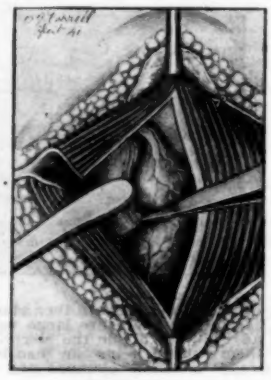

FIGURE XIII.—Firm retraction downwards and backwards is now needed so as to develop the upper pole and free it from the fascial covering. The gland is pushed medially by the closed dissecting forceps, while the blunt dissector pushes the fascia away from under the upper pole.

situated one comes to the sterno-mastoid fascia, which is carried across the mid-line of the neck. Beneath this lie the sterno-hyoid and omo-hyoid muscles, which are in the same morphological plane and are covered by the sterno-omo-hyoid fascia. Beneath these muscles lies the sterno-thyreoid muscle with its fascia forming a third plane.

FIGURE XIV.—The fascial layer is then pushed from the supero-medial portion of the upper pole by the blunt dissector, which usually slips down along the medial surface of the gland and can be worked gently under the upper pole, the fascia being pushed laterally as illustrated. The usual mistake is to attempt this separation too close to the isthmus. In many cases a large, retrolaryngeal projection of the postero-medial part of the gland may make this manœuvre difficult; but the secret is to work gently and always towards the tip of the upper pole of the gland, so as not to force the dissector through gland substance. At times it is helpful to push some gauze into or through this space and to develop the vascular leash by its use.

These points can be understood by reference to the two drawings (Diagrams I and II) made by Dr. Deane Butcher whilst he was attached to the surgical unit at the Prince Henry Hospital.

These three layers of fascia may fuse in the mid-line; but this depends on the amount of divarication of the infrahyoid muscles and on the number and complexity of the anterior jugular veins which in this region run between the sterno-mastoid and sterno-omo-hyoid layers of fascia. The sterno-omo-hyoid fascia runs backwards to join the postero-lateral part of the carotid sheath, while the sterno-thyreoid fascia runs onto the antero-medial part.

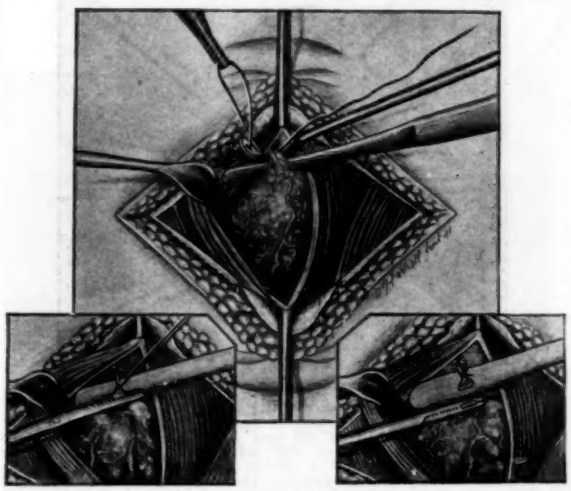

FIGURE XV. FIGURE XVI. FIGURE XVII.

FIGURE XV.—The blunt dissector can then be pushed under the superior thyreoid vessels and a clear space made for the passage of a ligature.

FIGURE XVI:—The first ligature is tied and held by the first assistant until a second ligature is tied some three millimetres above it. The gland is then crushed below the first ligature and held by artery forceps.

FIGURE XVII.—Both ligatures are cut short before the upper pole is divided so as to prevent an energetic assistant from pulling them off or tearing the vessels, which are often friable in elderly subjects.

The thyreoid gland itself is covered entirely by a fascial envelope, which is formed mainly by the deep layer of the sterno-thyreoid fascia, but medially contacts the fascial sheath of the œsophagus and then that of the trachea, whilst above the isthmus it makes contact with the fascia over the crico-thyreoid muscles.

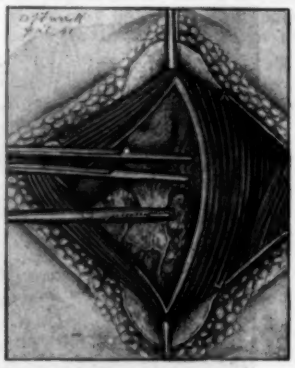

FIGURE XVIII.—The upper pole is then turned downwards and the medial aspect cleared by gauze dissection. The fascia running along the isthmus with the blood vessels in it can be doubly clamped by artery forceps and divided. It is convenient now to tie off the upper one of these forceps.

This fascial envelope of each lobe of the thyreoid gland is pear-shaped; the superior thyreoid vessels pierce it at the thin upper pole, the inferior thyreoid artery and middle thyreoid vein go through on the postero-lateral

FIGURE XIX.—The upper pole is next covered with a layer of gauze and seized in the surgeon's left hand. The gland is then lifted forward and turned medially. With the right hand the fascia is swept back from the gland substance by gauze, special attention being paid to the supero-medial border; this is the danger spot for possible injury to the superior parathyreoid and the inferior laryngeal nerve, which turns forwards here to go in under the inferior constrictor.

FIGURE XX.—As the fascial covering is pushed from the gland the lower pole can be elevated. The middle thyreoid vein is then cleared, ligated and divided. This immediately allows the fascia to be cleared well under the gland towards the hilum.

aspect and the inferior thyreoid veins pierce it at its lower extremity, while the two sides are connected across the region of the isthmus.

The parathyreoids, the external and inferior laryngeal nerves and the thymus or any nodules of this gland left in the pathway of its descent from neck to thorax are in different morphological planes from the thyreoid gland

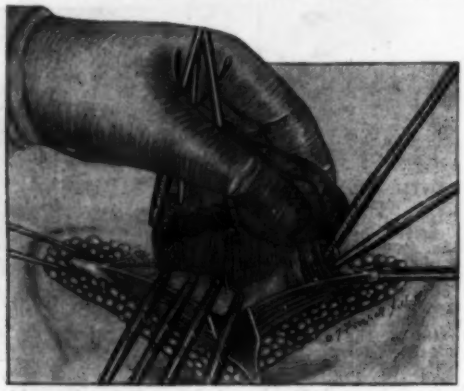

FIGURE XXI.—As the branches of the inferior thyreoid artery are seen they are seized in artery forceps and divided as they come through the fascia. The inferior thyreoid veins can also be clamped and divided. The fascial covering of the trachea is now exposed, and the gland should be freed from it by blunt dissection so that the trachea is not skinned.

FIGURE XXII.—The gland is now lifted up and freed from the tracheal fascia; it is necessary to use the back of the knife and to keep close to the gland. This freeing of the gland from the trachea is carried well over the mid-line so that the right lobe will lie over to the left side. The surgeon and first assistant then change places; but the second assistant still holds the upper Allis forceps and the retractor until the vessels are tied off. A gauze swab is then tucked into the bed of the thyreoid under the infrahyoid muscles, and the second assistant changes over to the left side. The loosened right lobe is then slightly drawn over to the right side by the first assistant, as this facilitates the application of Stages X to XX (shown in Figures X to XX) for the left lobe of the gland, if the standard subtotal resection has been decided upon. If a pyramidal process is present, this is now freed from its fascial covering, and its upper pole and any vessels are ligated. Once the left upper pole has been freed with any retrolaryngeal projection and the full extent of the left lobe has been determined, anchor forceps are clipped onto the gland capsule to determine the amount of gland to be saved. A portion about three centimetres by one centimetre is ample, and it is wiser to take too much than to leave too much.

and so lie outside the enveloping fascial capsule of the gland. This can be demonstrated in as early a stage of development as the twenty-millimetre embryo, and Weller in his excellent work refers to this fact. This point is stressed, since upon it depends the success of the technique now employed.

It will thus be seen that not only in theory, but in practice, it is possible to shell out the thyreoid gland from within its fascial envelope without damage to the important structures lying near by, once the vascular connexions of the gland have been divided within the fascial covering of the gland. This holds good also for any abnormal enlargements of the gland or for intrathoracic extensions,

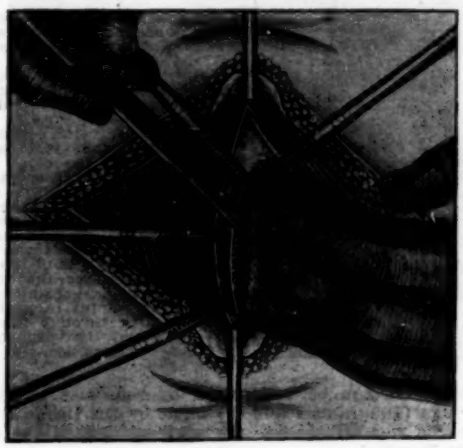

FIGURE XXIII.—The right lobe is then held in the surgeon's left hand and tensed over to the left. The gland capsule on the medial part of the left lobe is incised in a line close to the anchor forceps. Then, in order to excise a wedge and render subsequent suturing easier, the knife is made to cut into gland substance approximately parallel to its medial border and almost at right angles to the incision through the capsule. Small arteries close to the capsule are caught in forceps.

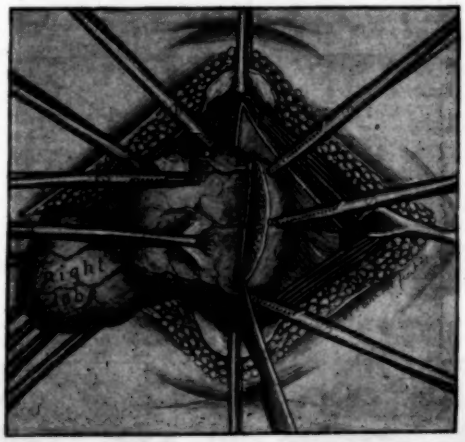

FIGURE XXIV.—The right lobe is then turned over to the right side, the left lobe is lifted up, and a similar cut is made into the postero-lateral part of the left lobe, joining the other incision above and below; a wedge is thus cut down into the substance of the gland, freeing the right lobe, isthmus and major part of the left lobe. Ligate any large arteries which may have been divided.

as these merely push the fascia ahead of them as they develop. The great importance of keeping within the fascial envelope thus becomes evident, and in difficult cases or when there is any doubt, no harm can be done so long as one works as close as possible to the true gland capsule or even within it on occasions.

The Danger Points.

The main danger points during the course of the operation are found along the postero-medial border of the

upper pole, as the superior parathyreoid lies just deep to the fascial envelope in this region; furthermore, this is the point where the inferior laryngeal nerve turns slightly forwards to go under the lower edge of the inferior constrictor muscle. In only 5% of cases does the superior parathyreoid lie at all lateral to the upper pole, but is always separated from it by the fascia. The other danger point for the inferior laryngeal nerve is just below the entry of the branches of the inferior thyreoid artery, since when the gland is turned medially the tissues are

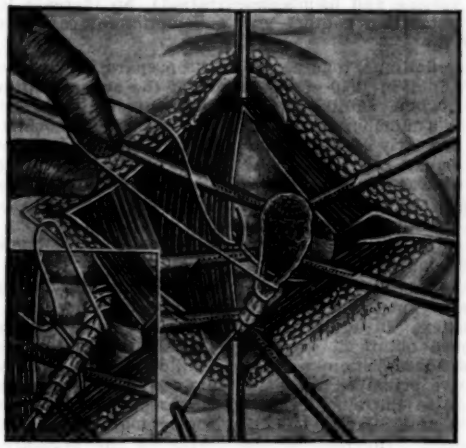

FIGURE XXV.—A fine curved needle with number 1 catgut is used to suture the cut surfaces together, a fairly deep "bite" being taken in order to secure approximation and control bleeding. Start from below, work upwards and then come back again; but now suture obliquely through the other stitches and closer to the cut edge, thus securing apposition and completing the hæmostasis (see inset).

With strict attention to the anatomical detail of the fascial planes, no damage should accrue to the parathyreoids; it is now many years since I have met with other than transitory tetany, and then only at rare intervals in a large series of cases, owing probably to temporary disturbance of circulation. In about 0·5% of cases or less one does divide the inferior laryngeal nerve, owing to faulty technique, but in some 5% there may be some physiological upset caused by undue tension during the operation and this disappears in a few weeks' time.

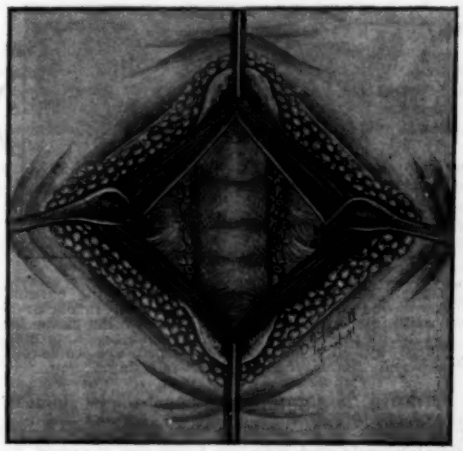

FIGURE XXVI.—If it is decided to perform total thyreoidectomy, carry on Stages XXI and XXII as described for the right lobe in Figures XXI and XXII until the whole gland can be removed. The surgeon and first assistant now change places and tie off all vessels, after which the second assistant comes to the right side again. After hæmostasis has been checked the thyreoid bed will be seen as now illustrated and with a complete fascial lining. It is seldom that the parathyreoids or inferior laryngeal nerve are seen, as they lie deep to the enveloping fascia of the thyreoid gland.

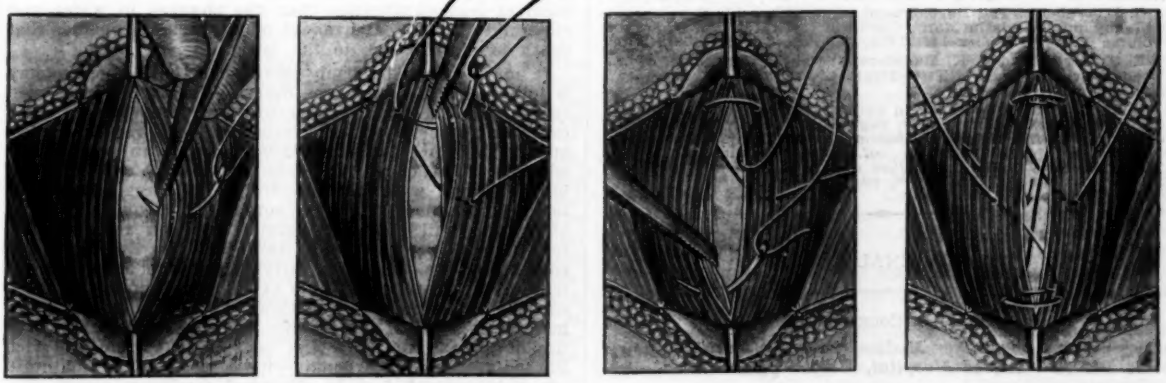

FIGURE XXVII. FIGURE XXVIII. FIGURE XXIX. FIGURE XXX.

FIGURES XXVII TO XXX.—The infrahyoid muscles are now drawn together by a figure-of-eight stitch as illustrated. If any drainage is required a small piece of corrugated rubber is placed on either side of the trachea and brought through the centre of the wound. If the infrahyoid muscles have been divided, it is better to bring the drain out deep to these muscles over the anterior border of the sterno-mastoid to the lateral end of the wound.

put on the stretch, and unless great care is exercised to see and secure the branches of the artery as they just enter the gland substance, it is easy to cut through the fascial envelope and penetrate into the region of the nerve.

The inferior parathyreoids usually lie well down out of harm's way, and in only 5% of cases are they at all closely related to the thyreoid gland; but in all cases there is the intervening layer of fascia, so it is imperative that the surgeon keep within the fascia when removing the thyreoid gland.

Difficulties.

The main difficulties met with have been in cases in which the following factors were at work: (i) prolonged or excessive iodine medication has caused iodine thyreoiditis and adhesion of the gland to its fascial envelope; (ii) a previous operation has been carried out without due observance of the fascial planes; (iii) old-technique deep X-ray therapy or prolonged "superficial" X-ray therapy has been used; (iv) carcinoma of the thyreoid gland has been present, extending beyond the true gland capsule;

(v) some acute or chronic infective process has been present in the gland, including Riedel's struma; (vi) the gland has been grossly enlarged. In such circumstances it may be advisable to divide the infrahyoid muscles after clamping them with Kocher's forceps, so as to allow of greater ease of access to the deeper parts of the gland. On rare occasions excessive hæmorrhage may determine one to abandon the operation rather than risk the patient's life, or else to be content with a hemithyreoidectomy and depend on control of the other lobe by deep X-ray therapy.

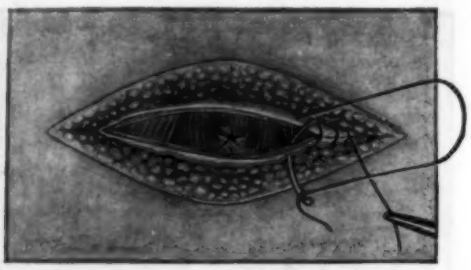

FIGURE XXXI.—A running stitch of fine catgut approximates the cut edges of the deep fascia and possibly platysma, but subcutaneous fatty tissue is not to be included. The skin edges are approximated by clips or stitches, which are removed not later than the fourth day.

Suture Material.

For ligatures it is best to use a number 0 silk for superficial vessels and number 1 for the thyreoid arteries and veins; but all silk ligatures must be cut very short. For suturing the gland substance, the muscles and the sterno-mastoid fascia number 1 catgut is used. With this technique serum seldom forms in the wound, and if strict asepsis is adhered to, extrusion of the silk knots seldom occurs.

Bibliography.

E. H. Norris: "Contributions to Embryology", Carnegie Institute of Washington, Volume XXVI, Number 159, page 249.

G. L. Weller: "The Development of the Thyroid, Parathyroid, and Thymus Glands in Man", Carnegie Institute of Washington, Volume XXIV, Number 141, 1933, page 95.

E. S. Meyers and R. K. Macpherson: "The Arrangement of the Deep Cervical Fascia", THE MEDICAL JOURNAL OF AUSTRALIA, November 12, 1938, page 813.

H. R. G. Poate: "Anatomy in Surgery", THE MEDICAL JOURNAL OF AUSTRALIA, October 7, 1939, page 534; "The Present Position of Surgery of the Thyreoid", *ibidem*, October 15, 1938, page 635; "Indication for, Technique of, and Results of Total Thyreoidectomy", *The Australian and New Zealand Journal of Surgery*, Volume X, July, 1940, page 48.

THE ADRENAL CORTEX.[1]

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Relationship of the Adrenal Cortex to Carbohydrate Metabolism.

EXTIRPATION experiments have shown that the adrenal cortex, but not the medulla, is essential for life. Clinical observations support this view. Thus in cases of Addison's disease post-mortem examinations have revealed almost complete absence of cortical tissue, whilst staining reactions indicate no deficiency of adrenaline. In cats and dogs experimental adrenal insufficiency is a well-defined phenomenon. Removal of both glands at one operation results in acute symptoms, followed by death in approximately thirty-six hours. After a two-stage operation with an interval of seven to ten days, a more chronic condition ensues and death occurs within six to fourteen days. The most characteristic symptoms are anorexia, muscular weakness, spastic gait, loss of weight and lessened resistance to infections. Important biochemical disturbances are produced—a rise in the blood urea and non-protein nitrogen content, a decrease in the plasma bicarbonate content, an increased excretion of sodium and water by the kidneys with loss of fluid from tissues and blood (the last-mentioned leading to decrease in blood volume and hæmoconcentration), an increase in the blood potassium content and a depletion of carbohydrate reserves.

These findings, together with the preparation by Swingle and Pfiffner in 1930 of an extract of the suprarenal cortex which, when administered to adrenalectomized animals even in a moribund state, restored them to a normal condition, clearly demonstrated the hormonal function of the cortex. In these early investigations the name "cortin" was given to the hormone of the adrenal cortex, and all the physiological effects were ascribed to the "vital" hormone of the gland. "Cortin" activity was assessed by ability to maintain a normal blood composition in dogs deprived of their adrenal glands.

In the last few years a number of crystalline compounds possessing physiological activity have been isolated from the cortex. In addition a purified amorphous concentrate has been obtained, extremely potent in maintaining the life of adrenalectomized dogs. Following the isolation of an active principle, a considerable amount of research was devoted to the elucidation of the precise mode of action. Britton and Silvette have vigorously maintained that the "prepotent" function of the cortex is the regulation of carbohydrate metabolism. Several other groups of workers consider that regulation of the circulation, plasma electrolytes and water metabolism are of primary importance. In addition, the question has arisen as to whether the adrenal cortex elaborates one hormone, which can be regarded as the "vital" hormone, or whether a number of hormones are involved, each with a specific function.

Admittedly disturbances in carbohydrate metabolism occur in adrenalectomized animals; but if anything, they seem of less significance than the changes in water and electrolyte balance, and indeed many workers consider that death is directly due to the latter.

It now seems to be firmly established that adrenalectomy is followed by a pronounced decrease in the liver glycogen content. Long and his collaborators[(1)] have made some important observations on adrenalectomized rats and mice, and have shown that in these species an appropriate administration of sodium salts maintains not only life, but a normal water and electrolyte balance. Adrenalectomized animals given a diet containing adequate amounts of sodium will maintain normal glycogen reserves. If food is withdrawn, a rapid decline in the liver glycogen content follows, and after forty-eight hours hypoglycæmia may occur. Apparently there exists a relationship between carbohydrate metabolism, electrolyte distribution and water metabolism. With non-specific therapy—for example, the use of glucose and of a diet poor in potassium and rich in sodium and bicarbonate—it is possible to maintain adrenalectomized dogs in a normal condition, except that they do not possess the ability to withstand stress. If the administration of sodium chloride is stopped even for a short time, acute cortical insufficiency ensues. Clinically, in this connexion these findings agree with the fact that patients suffering from Addison's disease may be maintained in an apparently normal state by the administration of large quantities of sodium chloride; but it would seem that the question of resistance to stress should be kept in mind.

The crystalline compounds having "cortin"-like activity are sterols, and in order to appreciate their physiological actions it is necessary to refer briefly to the individual constitution of some of these compounds. The sterol nucleus with the conventional numbering of the carbon atoms is shown in Figure I.

[1] A summary of a paper read at a meeting of the Alfred Hospital Clinical Society.

The following are the most important physiologically active compounds that have been isolated: (i) corticosterone, (ii) desoxycorticosterone, (iii) dehydrocorticosterone, (iv) compound E. The constitution of corticosterone is shown in Figure II. All four compounds have the following in common: (i) a keto group (−C = O) at C_3; (ii) a double bond at C_4—C_5; (iii) a side chain at C_{17}.

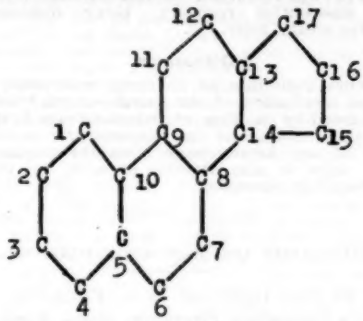

FIGURE I.

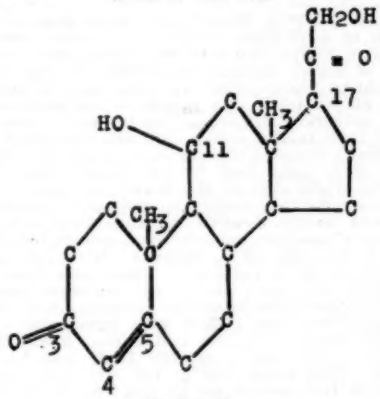

FIGURE II.

The other compounds differ from corticosterone in the following ways: (i) Desoxycorticosterone does not possess a −OH group at C_{11}. (ii) Dehydrocorticosterone has an oxygen atom instead of −OH at C_{11}. (iii) Compound E has a −OH group at C_{17} and an oxygen atom at C_{11}.

Desoxycorticosterone, isolated by Reichstein in 1938, is the most active crystalline compound for maintaining a normal blood chemistry in dogs subjected to adrenalectomy. Administered to normal animals, it increases the serum sodium content and decreases the serum potassium content, the latter sometimes to such an extent that paralysis occurs. Progesterone, the *corpus luteum* hormone, is closely related to desoxycorticosterone, the only difference being an oxygen atom less in the side chain at C_{17}. It is amazing that such a slight difference in constitution can produce such divergent physiological actions, although it has been shown that large doses of progesterone have a slight "cortin" activity. Experiments on rats by Long and Katzin clearly indicate that the adrenal cortex is implicated in carbohydrate metabolism. Large doses of cortical extract were administered to fasting normal rats, and a great increase was found in liver glycogen content, as judged from values in untreated controls. No changes in muscle glycogen content were noted. The increase in urinary nitrogen excretion suggested that the source of the new carbohydrate was protein.

In our own experiments with young rabbits we have been able to confirm these results. "Eschatin", a preparation of Parke, Davis and Company, containing the water-soluble constituents, caused a pronounced deposition of glycogen in the liver. On the other hand, desoxycorticosterone, most potent in respect of maintaining a normal electrolyte balance, was quite inactive. Other preparations were not available for our studies; but recently Grattan and Jensen[2] have shown that corticosterone and 17 hydroxy-11-dehydrocorticosterone promote the deposition of glycogen in the liver in rats. Desoxycorticosterone under the same conditions had no influence. In addition, we have found that progesterone can cause a deposition of glycogen in the liver of young rabbits. Testosterone, œstrin and anhydro-hydroxyprogesterone were inactive. The compounds that caused an increase in liver glycogen content had no influence on liver fat content.

Traumatic Shock and the Suprarenal Cortex.

Although a close similarity exists between the physiological disturbances produced by wound and histamine shock, with one exception, namely, extensive burns, it has not been possible to demonstrate histamine or other depressor substances in the blood of patients or animals suffering from shock.

Recently Seyle[3] has presented experimental evidence suggesting a connexion between the adrenal cortex and the phenomena of shock. Groups of rats were used and the gut was traumatized by the application of clamps. Animals treated with corticosterone, in contrast to untreated controls, did not suffer from shock. Tested under similar conditions, desoxycorticosterone and a commercial cortical extract were relatively ineffective. Seyle thinks that the failure of the latter to prevent shock lies in the fact that it does not contain sufficient quantities of the active steroid and in addition has "harmful contaminating" substances.

Extremely relevant to this work is an editorial in *The Journal of the American Medical Association*[4] on the therapeutic applications of desoxycorticosterone. A commercial firm is strongly criticized for advocating the use of this compound in an indiscriminate fashion. It is pointed out that the clinical evidence of the value of desoxycorticosterone in surgical shock is scanty. From Seyle's experiments described above it would also appear that such treatment is unsound.

Unfortunately commercial supplies of corticosterone are unobtainable in this country; but it would appear worth while to carry out a large-scale clinical investigation with this compound.

Before I leave this problem, there is another possibility to be considered. Freeman[5] has pointed out that all the factors known to produce shock have one physiological action in common—namely, they all produce hyperactivity of the sympathetic nervous system—and in 1922 Rapport[6] reported an increased output of adrenaline in shock. Since shock is characterized by reduction in circulating blood volume, it occurred to Freeman to test the effect of experimental over-activity of the sympathetic nervous system on blood volume. Cats under "Dial" anæsthesia were given a continuous infusion of adrenaline at the rate of 0·006 milligramme per minute per kilogram of body weight. This treatment was found to produce a decrease in blood volume which did not occur if the vasoconstrictor effects of adrenaline were inhibited by a previous injection of ergotoxine.

On the clinical side there is as yet insufficient evidence to correlate sympathetic over-activity with the production of surgical shock; but Walter,[7] in a study of the blood changes in a series of 50 patients undergoing abdominal operations, found in one patient, exhibiting symptoms of shock, an increased adrenaline content of the blood. Unfortunately this was the only case in which the amount of adrenaline in the blood was estimated.

In agreement with the above findings, other workers have been able to produce experimental shock by the intravenous injection of large doses of adrenaline.

The precise physiology of shock still remains unsolved; but the influence of corticosterone and sympathetic over-activity requires further investigation.

References.

[1] C. N. H. Long, B. Katzin and E. G. Fry: "The Adrenal Cortex and Carbohydrate Metabolism", *Endocrinology*, Volume XXVI, 1940, page 309.

(42) J. F. Grattan and H. Jensen: "The Effect of the Pituitary Adrenocorticotropic Hormone and of Various Adrenal Cortical Principles on Insulin Hypoglycemia and Liver Glycogen", *The Journal of Biological Chemistry*, Volume CXXXV, 1940, page 511.
(43) H. Seyle and C. Dosne: "Treatment of Wound Shock with Corticosterone", *The Lancet*, Volume II, July 20, 1940, page 70.
(44) "Desoxycorticosterone Acetate", *The Journal of the American Medical Association*, Volume CXV, 1940, page 47.
(45) N. E. Freeman: "Decrease in Blood Volume after Prolonged Hyperactivity of the Sympathetic Nervous System", *The American Journal of Physiology*, Volume CIII, 1933, page 185.
(46) D. Rapport: Quoted by N. E. Freeman, *loco citato*.
(47) W. W. Walther: "Blood Changes after Surgical Operations", *The Lancet*, Volume I, January 2, 1937, page 6.

Reports of Cases.

A CASE OF PRIMARY PERITONEAL PREGNANCY.

By CLIVE WILLIAMS, M.B., B.S.,
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THE following case is of interest owing to the rarity of the condition and the doubt that has existed as to the possibility that such a condition could arise.

Clinical Record.

J.W., aged twenty-three years, a single woman, suffered a sudden collapse on the evening of June 13, and on examination was found to be *in extremis*; her pulse was imperceptible and she was incapable of answering any questions. "Coramine" (0·5 cubic centimetre), given subcutaneously, revived her sufficiently for the following history to be extracted. After a period of amenorrhœa lasting for fourteen days, during which time, on the assumption that a pregnancy had occurred, various tablets had been taken in the attempt to procure an abortion; hæmorrhage had occurred *per vaginam*, accompanied by abdominal pain. The discharge was bright in colour, and the hæmorrhage lasted intermittently for about three days. At no time was there any history of anything approaching a discharge dark in colour. During the ensuing fourteen days she suffered intermittent abdominal pain, mostly located in the left iliac region and radiating down the left leg; the pain was unaccompanied by any vaginal bleeding. This pain during the last few days was accompanied by a feeling of faintness, and in addition by a pain in her right shoulder. They were of increasing severity, and eventually culminated in the severe attack which caused her final collapse.

On examination she was obviously in a grave condition, with an almost imperceptible pulse, and showing the signs of extreme hæmorrhage. A tense, extremely tender swelling occupied the left iliac fossa. Vaginal examination disclosed a tense, tender, fluctuant swelling filling the left fornix and extending above the umbilical level. In addition there was a small indurated area in the vaginal wall about two centimetres to the left and slightly behind the cervix. This rather suggested a wound of the vaginal vault, and inquiries were made as to the use or otherwise of an instrument to procure an abortion. This was denied, however.

A diagnosis of ruptured tubal pregnancy was made and the patient was admitted to hospital for immediate operation. An intravenous infusion of 5% glucose and saline solution was begun, and this was continued during the subsequent operation and for several hours afterwards.

Under ethyl chloride and ether anæsthesia the abdomen was opened by a mid-line incision and was found to be full of blood and blood clot. On the assumption that the condition was a left-sided tubal pregnancy, the left tube and ovary were rapidly delivered. Apart from a hyperæmic condition of the surface of the left ovary, no abnormality was present. The right tube and ovary were likewise normal and showed no signs of a tubal pregnancy. At this stage of the operation it was noticed that bright blood was welling up from Douglas's pouch in rather an alarming quantity. In the cleaning out of the blood clot, which was packed tightly in Douglas's pouch, a fœtus of about the size of a four weeks' pregnancy was delivered. It was then noted that bright blood was coming from a spot on the floor of Douglas's pouch, somewhat to the left of the mid-line, and that the placental tissues were still *in situ*. These were rapidly stripped from the peritoneal surface; a shallow crater was left, about two and a half centimetres (one inch) in diameter, to the left of the mid-line and slightly behind the level of the cervix, and corresponding in position to the indurated area felt in the vault of the vagina previously mentioned. A large artery was bleeding profusely from the anterior portion of this cavity (probably a cervical branch of the uterine artery), and this was clamped and ligated and the crater-like cavity was oversewn with catgut. The anterior surface of the rectum was eroded for an area about one centimetre in diameter, and this was also oversewn.

The patient was returned to bed in fair condition and made an uneventful recovery, being discharged from hospital three weeks later.

Discussion.

The patient's condition at no stage warranted a detailed study of the mechanics of the pseudo-uterus; but this was evidently formed by the floor of Douglas's pouch, the anterior surface of the rectum and the left ovary.

The lack of any hæmorrhage from the vagina after the initial few days is also worthy of note, in view of the condition found at operation.

TUBERCULOSIS OF THE CERVICAL GLANDS.

By GUY GRIFFITHS, M.D., F.R.A.C.P.,
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Clinical Record.

ON August 26, 1924, Miss E.M.C., aged sixty-three years, complained of a painless lump in the neck; she had had many similar lumps and had undergone six operations at the hands of a very distinguished surgeon. The first operation was thirty years earlier, in 1894, and the last in 1920; they were all for tubercular glands of the neck; she feared a seventh operation and wished to try other treatment.

She was then bright, active and well nourished, but pale. She exhibited a rounded thoracic kyphosis with some rigidity, and there were some four or five sound, linear, operative scars on the right side of the neck; her mouth was awry and she protruded her tongue to the left. Above the right clavicle, and partly covered by the sterno-cleido-mastoid muscle, was a movable, smooth, oval, subcutaneous swelling, nearly half the size of a walnut. No large axillary or inguinal glands were palpable. There was some muscular rigidity along the upper half of the vertebral border of the right scapula, and here bronchial breathing was heard. The cardiac apex beat was in the fifth intercostal space, four and a half inches from the mid-sternal line. The mitral sounds were clear, but a systolic bruit was heard in the aortic area. The pulse rate at the left wrist was 108 per minute while the patient sat at rest; at the right wrist it was hardly palpable; she said it had always been so. No abdominal abnormality was found.

On further observation her blood appeared of good colour and consistence; it flowed freely and coagulated readily; the red blood cells were of good number, shape and size, and formed good rouleaux. Her temperature range was normal—morning 97° F., evening 99° F. She weighed nine stone ten pounds fully clad. Her urine was pale, clear and acid, and contained neither albumin nor glucose.

Miss C. reacted to tuberculin. After subcutaneous injection of 0·0003 cubic centimetre of albumose-free tuberculin ("T.A.F.") there was a slight local reaction, and after the injection of 0·00045 cubic centimetre there was a larger one—a smooth, round, hard, red, hot, tender swelling in and beneath the skin of the arm at the site of the injection.

She was treated with fresh air, rest, gentle exercise, plain nourishing food, a mixture of citrate of iron and ammonia with iodide of potash and a little arsenic, and by injections of "T.A.F.", the doses increasing to 0·009 cubic centimetre. Her temperature once rose to 100° F. and she fainted, and once to 102° F. and she felt ill; but always the reactions soon abated, she felt better, the gland decreased in size, and she asked for more injections. Original old tuberculin ("T.O.A.") was given, from 0·001 cubic centimetre to 0·6 cubic centimetre; "T.A.F." was again given, from 0·0001 to 0·45 cubic centimetre; *Perlsucht* tuberculin ("P.T.") was given, from 0·001 to 0·26 cubic centimetre; and bacillary emulsion ("B.E.") was given, from 0·02 to 0·05 cubic centimetre.

The patient had intervals without injections, but found that the gland grew larger and others appeared in the left side of the neck, and so she returned for more. In the course of seventeen years she has had "P.T." up to 0·64 cubic centimetre, "T.A.F." up to 0·9 cubic centimetre, old tuberculin up to 0·8 cubic centimetre, and "P.T." again up to 0·55 cubic centimetre; in each case the amount named is that given in the last single dose.

The patient has suffered from the following intercurrent illnesses: conjunctivitis, for which she has been under the

care of ophthalmic surgeons; bronchitis, treated with the inhalation of dilute formaldehyde, with a simple linctus, with phenazone in aromatic spirit of ammonia, with the dilute liniment of turpentine and acetic acid, and with the fumes of creosote; *herpes supraorbitalis*, for which the late Dr. G. R. Hamilton saw her in consultation in 1932 (this illness necessitated a week in Saint Luke's Hospital, and her eye narrowly escaped); and dizzy "turns", with dim sight, slight irregularity of the pulse and accentuation of the second aortic sound, relieved after small doses of digitalis. In 1936 she was worried by hyperkeratoses of the left cheek, an acuminate lesion of the left external ear and a cutaneous horn; the lesions were treated by Dr. A. L. Dawson with the application of a radium plaque, diathermy and excision respectively.

In December, 1938, Miss C. was breathless, her fingers became numb and the second sound at the aortic cardiac area was much accentuated; her blood pressure was 145 millimetres of mercury systolic and 85 diastolic. An electrocardiographic examination was suggested, but she demurred. The pulse was regular; she rested and improved in health.

In January, 1939, she felt weak from the continued hot weather, and one night she had numbness in the left hand, dry retching and an agonizing pain in the breast and left arm, and was alarmingly ill. Dr. Jefferis relieved her pain by a hypodermic injection and found that her second aortic sound was accentuated and that her blood pressure was 130 millimetres of mercury systolic and 90 diastolic. He kept her in bed and gave one and a half grains of "Amytal" night and morning and fifteen grains of "Diuretin" three times a day; no trinitrin was given. I saw her with him and concurred in his findings, his diagnosis of coronary occlusion and his treatment. Her condition improved very rapidly under devoted and capable family care and expert nursing, and after three weeks in bed she was allowed up. There has been no recurrence of any cardiac attack; but the blood pressure has gradually risen to the following figures: 155 millimetres of mercury systolic and 90 diastolic in March, 1940, and 175 millimetres of mercury systolic and 90 diastolic in September. A little numbness in the right hand was sometimes present. The patient has suffered twice from bronchitis in a degree serious for a patient of her age, but otherwise has done well.

In September, 1940, the long-delayed electrocardiogram was made by Dr. A. J. Hood Stobo, who reported as follows:

> The rhythm is normal, as also are the auricular and ventricular complexes, and there is no delay in conduction in the bundle or myocardium. There is no definite electrocardiographic evidence of any coronary or myocardial defect.

Dr. H. R. Sear undertook a skiascopic and skiagraphic examination of the thorax, with the following findings: much osseous kyphosis; much atheroma of the aortic arch and some dilatation of the first part of the aorta, but no enlargement of the heart; poor diaphragmatic movements; chronic bronchitis; calcification of the hilar and right paratracheal glands and of the cervical glands above the right apex, but no evidence of active tuberculosis.

In July, 1941, Miss C. is bright, cheerful and active; she enjoys gentle exercise in her garden; she is stooped and a little pale, and sometimes coughs; slight malleolar œdema is present, and in her urine there is a trace of albumin, but no casts are seen; otherwise she is very well. The cervical glands are palpable only with difficulty, as also is the pulse at the right wrist. She is eighty years of age.

Summary and Comment.

A case is described of tuberculosis of the cervical lymphatic glands, recurring six times after expert surgical excision and then yielding to injections of tuberculin. It is suggested that the repeated invasions of tubercle bacilli into the cervical glands came from a reservoir in the pulmonary lymph glands and that the ultimate success was due to the specific action of the tuberculin in producing a curative fibrosis, both in the neck and in this deep-seated reservoir.

The patient's intercurrent maladies, one suggesting coronary occlusion perhaps from a tiny atheromatous embolus, and the happy issue are recorded.

Reviews.

BOXING.

E. JOKL's up-to-date boxing Baedeker offers a wide range of subject matter.[1] The consideration of pugilistic physiology and pathology is richly supplemented with case histories of cerebral concussion and other post-traumatic catastrophes, and many anecdotes of fistic fun and rough-house tricks are not omitted. An extensive boxing bibliography is included, to which the author has contributed sixteen publications. Many references are made to material published so recently as 1940. The discussion of the nature of cerebral concussion and unconsciousness is interesting if somewhat uncritical. Generalizations such as the following are too frequently encountered: "The writer has not the slightest doubt that chest injuries in boxers are considerably more common than is generally assumed" (page 53).

Medical jurists and educational authorities would be interested, and perhaps influenced, by the chapters devoted to those aspects of the subject matter. The author states that boxing is a "dangerous and therefore undesirable form of physical education", and concludes on page 222 with the following remark: "If this monograph will serve no other purpose than to immediately effect the elimination of boxing from the physical training programme of the armed forces, and more especially of the Air Force, it will have fulfilled an important task." This is one side of the story; the other comes from an Australian surgeon, who reports to us that in his twenty years of experience of amateur boxing, in schools, universities and the services, not one case of physically or mentally debilitating after-effect has been seen.

The book is well illustrated and would make an interesting addition to the shelves of a critical collector.

[1] "The Medical Aspect of Boxing", by E. Jokl, M.D.; 1941. Pretoria: J. L. van Schaik, Limited. Demy 8vo, pp. 251.

SURGERY OF MODERN WARFARE.

IN Part II of "Surgery of Modern Warfare"[1] McIndoe concludes his chapter upon skin grafting—the cutting of the grafts, the preparation of the bed, the application of the grafts and the post-operative treatment. The following chapter will furnish those interested in maggot therapy with every detail necessary for the successful breeding and employment of maggots.

Thoracic wounds are admirably dealt with by Tudor Edwards. He favours early evacuation with air replacement in the treatment of hæmothorax. The question of anæsthesia in major thoracic surgery is of great importance, and Magill's opinions will command respect. He prefers local and intravenous methods, holding that the value of positive pressure inhalational anæsthesia is debatable.

The names of those who contribute the chapters upon abdominal wounds—Charles, Fraser and Gordon-Taylor—are a sufficient indication of their authority and value. The importance of careful planning of the incision of access is stressed, particularly in relationship to wounds which probably involve the large bowel. In such wounds, perhaps more than in any other, the surgeon without a background of military surgical experience will welcome the helpful advice given in these pages, the fruits of a vast experience gained in the last war. The particular difficulties associated with wounds of the rectum and buttocks are in the capable hands of Gordon-Watson, whose special domain this has long been conceded to be. A concise chapter upon post-operative abdominal complications is devoted to certain more recent methods—chemotherapy *et cetera*—which may usefully be employed as adjuvants in the treatment of those complications which were only too well known to the last generation of casualty clearing station surgeons.

Wounds of the genito-urinary tract, except for their frequent association with other visceral injuries, present problems that are not strikingly dissimilar to those met in civil surgery. In certain perineal wounds, however, it seems likely that bladder drainage by the perineal route presents definite advantages.

The several chapters of Section IV are devoted to a consideration of wounds of blood vessels. If one chapter can be singled out for particular approbation, it is that dealing with the exposure of the main vessels of the limbs. It must be remembered that "classical approaches to the vessels of the limbs find little or no place in the surgery of the vascular injuries of war. Missiles do not inflict their damage conveniently at the 'seats of election' . . . and so it comes about that the standard restricted exposures are both dangerous and inadequate." Those who have recollections of past struggles with the posterior tibial, the gluteal and the subclavian arteries will appreciate the debt that surgery owes to the work of Fiolle and Delmas, to whom tribute is paid here. In dealing with wounds of arteries, Learmonth, in

[1] "Surgery of Modern Warfare", edited by H. Bailey, F.R.C.S. London: E. and S. Livingstone. Part II, 1940. Super royal 8vo, pp. 163, with illustrations. Price: 12s. 6d. net. Part III, 1941. Super royal 8vo, pp. 159, with illustrations. Price: 12s. 6d. net. Part IV, 1941. Super royal 8vo, pp 160, with illustrations and diagrams. Price: 12s. 6d. net.

describing the types of trauma, deals with the phenomenon, not unknown in civil surgery, of arterial stupor and its treatment. An excellent description of conservative methods follows, and it includes a discussion of the maintenance of nutrition of the affected limb. It is interesting to find that he preserves an open mind upon the controversial question of ligation of the accompanying vein when it becomes necessary to tie the common femoral or popliteal artery.

This Part II of "Surgery of Modern Warfare" in every way maintains the very high standard of subject matter, clarity of presentation and production that called for such unstinted approval of Part I.

In Part III there is found the continuation of the section on wounds of blood vessels, and the two chapters devoted here to the subject are of great interest and importance. The first, contributed by Matheson and Gordon Murray, deals with the use of heparin in conservative vascular surgery, particularly in conjunction with Tuffier's method of temporary arterial intubation. The next, by Waugh, on secondary hæmorrhage, is based upon a very large practical experience and provides most helpful advice in these anxious and difficult cases. The final chapter in this section deals with a problem almost peculiar to war surgery, that of arterio-venous communications.

Section V is concerned with the subjects of the injuries of peripheral nerves and of wounds involving tendons. Well illustrated and full of practical points, the section devoted to the former is, of necessity, somewhat condensed. The Bunnell-Mayer technique of tendon suture is well described by Mouat, who finds himself in agreement with Teece in his poor prognosis of flexor tendon sheath injuries. In such cases he advises the employment of a free tendon graft.

Section VI deals with methods of immobilizing the limbs—the application of extension, the use of the Thomas's splint and frame, of Braun's splint, of Cramer wire, and, last but not least, of plaster of Paris. McMurray writes as the doughty champion of the Liverpool school, ably defending the use of skin extension and the Thomas's bed knee splint in a variety of injuries. The description of the proper method of application and use of this splint is full and clear, and emphasis is laid upon those important points the omission of which leads to an imperfect result. When skeletal traction becomes necessary McMurray prefers the Steinmann nail, with swivel stirrup, to Kirschner's wire. A useful chapter upon the use of Braun's splint and its modifications is contributed by Lloyd. Here the attention to detail which is characteristic of the whole work is evident in the instructions for the preparation and application of this splint. The use of Cramer wire as a temporary method of splintage is well illustrated. The section concludes with some 25 pages devoted to the modern application of plaster of Paris. This is well illustrated and eminently practical, the dangers and complications that may occur following plaster fixation being very wisely stressed. This chapter, as well as those that follow it upon wounds of the hand and of the foot, may well be studied by those engaged in the surgery of civil trauma. The influence of the teachings of Kanavel and of Iselin is evident in the important subject of infections of the hand. The part under review concludes with chapters upon wounds of the knee and hip joints. It would seem, in view of the great frequency and importance of the former, that more space might have been given to consideration of these injuries and their sequelæ, for this penultimate chapter leaves the reader, so far keyed to a high pitch of anticipation, with some feeling of frustration.

Wounds of joints receive further consideration in the first two chapters of Part IV. Here, too, in each case a description of the surgical anatomy of the part precedes the description of the surgical procedure and problems involved in the treatment of both recent and late wounds. The methods of immobilization to be employed and other considerations peculiar to individual lesions are included. Finochietto's method of stirrup traction in wounds of the ankle joint is mentioned only to be condemned, but the bad results spoken of are surely not of frequent occurrence.

The twenty pages that follow are devoted to the enormously important subject of wounds of bone, largely, as is pointed out by the writer, Robert Milne, that of compound fracture. He does well to emphasize that ". . . sound judgement—when to operate, when to amputate, when to excise the wound meticulously, and when to perform rapid but purposeful debridement—is far more important than blowing sulphonamide powder around the broken bone and encasing the limb in plaster . . ." The factors which influence the decision to amputate and other general considerations common to all such injuries are very well set out, as are the features of individual types of injury. The method of operative approach to compound fractures is fully detailed, and such controversial matters as Leriche's esquillectomy, the use of antiseptics in the wound, and its closure, are discussed in relation to the earlier type of case. Later cases receive separate consideration. Mention is made of the use of brachial plexus block in upper limb injuries, but the suggested amount of anæsthetic solution is possibly inadequate for general use.

Several very useful pages are given up to the subjects of sequestrum formation, sequestrectomy and the treatment of bone cavities. All these loom large to those who are concerned with the later care of bone injuries of this type, which in the case of bone cavities often demand a considerable experience of general plastic methods. In this connexion it would seem that the *tulle gras* method of Thiersch grafting described presents difficulties readily overcome by the direct application of the grafts to the mould of dental wax.

A thoroughly practical chapter upon amputations is contributed by de Courcy Wheeler. It is followed by one upon the same subject by Verrall, who writes from the viewpoint of the fitting of the artificial limb. He defends the guillotine amputation in selected cases and describes the subsequent skin traction, too often neglected. Both these authorities speak from a very wide experience, and their contributions will be read with the greatest interest. It is evident that there is still room for conflicting views in certain directions—the optimum length of the below-knee stump, retention of the fibular head in such amputations, the sectioning of main nerve trunks, and the merits or otherwise of Syme's amputation.

Section IX deals in the first chapter with wounds of the face and jaws, and is contributed by Kilner. In it he outlines treatment designed to ensure that "nothing will be done to jeopardise the chances of obtaining a good final result while making certain that everything possible is done to prepare the way for later reconstruction", of which he indicates briefly the main types. The arrangement of the text is admirable, dividing wounds of this region into those of the covering, supporting (skeletal) and lining elements, alone or in varying combinations, with or without loss of tissue. For those newly introduced to the very special problems presented by these injuries order is thus made to emerge out of confusion. The author disclaims any intention of more than outlining methods, but in this he is most successful, dealing not only with facial deformities involving reconstruction, but with fractures of mandible and maxilla, zygoma and nose, with or without skeletal defect. An even more specialized and difficult field, that of the eyelids and orbit, is most thoroughly traversed by Stallard.

PHYSIOLOGY FOR BEGINNERS.

THE publication in Australia of a short book by Dr. A. A. Abbie, comprising the essentials of physiology and anatomy, will be welcomed by many who have felt the need for such a book.[1] We are informed in the preface that the work covers the ground for physiology examinations up to and including that for general nurses; the book cover also states that there is an adequate summary of human structure to clarify the physiological discussions for those who are not taking a concurrent course in human anatomy. The book obviously must be judged on these statements, and we have no hesitation in stating that the author has produced a book that will meet the needs of nurses and other students to whom an intimate and profound knowledge of the subject is not necessary. Any book intended for nurses will be charged with sins of omission or of unnecessary inclusion; very few persons will agree on just how much a nurse should be expected to know. Criticism of this kind may possibly be levelled at Dr. Abbie's choice of material; but if this does occur, most persons will regard the criticism as captious. The matter has been wisely chosen and the first reaction of the reader is one of admiration that so much important material has been included, while the text is still very readable. The numerous diagrams that illustrate the text are from Dr. Abbie's own pen, and make for clarity. Special attention should, we think, be drawn to a valuable chapter on certain aspects of the fundamental sciences. Here in a few words are set out the essential facts on such subjects as heat, sound, light, electricity, inorganic and organic chemical substances, diffusion, osmosis and so on. To object to this chapter would be ridiculous. Some persons may hold that the chapter on chromosomes, sex determination, heredity and evolution is unnecessary; we would be inclined to agree, but would not regard our objection as very important. We trust that this book will have the large sale that it deserves.

[1] "Human Physiology", by A. A. Abbie, M.D., D.Sc., Ph.D.; 1941. Sydney: Angus and Robertson Limited. Crown 8vo, pp. 336, with 74 illustrations. Price: 6s. net.

The Medical Journal of Australia

SATURDAY, SEPTEMBER 20, 1941.

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THYREOTOXICOSIS.

WHEN a clinician sets out to treat a patient who is suffering from a disease, however well known and clearly defined the clinical picture may be, he does not say to himself: "Here is a case of 'so-and-so' and my mental index tells me that the treatment for this disease is such-and-such a procedure or remedy." He knows that this method of attack has little to commend it and will neither achieve the best result for his patient nor add to his own store of knowledge. He tries to base both his diagnosis and his treatment on an understanding of the morbid process that has given rise to the clinical picture; in other words, he keeps pathogenesis always before his mind. The man who would use his mental index of treatment without trying to recall what the disease really is and how it arises, belongs to the type of clinician who would read a tome labelled "Index of Treatment" and never open a book on pathology, nosology, symptomatology, and so on. Such a method of approach, apart from being futile, makes its user little more than an automaton and may start him on the road to mental atrophy. The condition known as thyreotoxicosis is one about which an extensive literature has gathered; here are some well-established facts, much theory that appears to be justified on the basis of those facts, and a great deal more that is frank hypothesis. The subject is as fascinating as it is enormous, and this very fascination is a snare for the unwary, tempting him to build on an untried and insecure foundation. A recent meeting of the New South Wales Branch of the British Medical Association was concerned with the treatment of thyreotoxicosis, and the three papers read at that meeting are published in this issue. No doubt the extent of the ground to be covered by such a discussion was the reason why no separate paper was devoted to pathogenesis; but readers will note that the authors of the papers did not confine their remarks solely to treatment and that the opener of the discussion, Dr. F. B. Byrom, dealt shortly but effectively with several vital points in the pathology of the condition. We venture to assert that if practitioners will read carefully the papers by Dr. S. A. Smith, Dr. H. R. G. Poate and Dr. Charles De Monchaux, they will be helped in the clinical approach to a patient with thyreotoxicosis and, if they have not become so before, they will, like all others who have studied the subject, be far from content with the present state of our knowledge about it.

The most interesting questions in the pathogenesis of thyreotoxicosis, and possibly the most important in view of possible developments in the future, have to do with the relationship between the thyreoid gland and the anterior lobe of the pituitary body, the thyreotropic hormone and the possible isolation in the future of an effective antihormone. Dr. S. A. Smith made this clear in his opening remarks. Later on, Dr. Byrom stated the blunt facts, first, that though an antihormone to the thyreotropic hormone of the anterior pituitary was known, it could not as yet be used in treatment, and secondly, that there was no evidence for or against the view that the pituitary was implicated in thyreotoxicosis. It is the work which has made this statement possible, that has created much of the fascination surrounding the whole subject. The clinician does not need to be reminded that the normal function and structure of the thyreoid gland are dependent on an adequate exogenous supply of iodine and that the thyreoid has in point of fact a specific affinity for iodine, which means that it seizes upon practically all the iodine that normally gains access to the body. Dr. Smith has reminded us that the iodine needs of the normal body are small. Those who wish to read a useful account of the physiology of the thyreoid gland are referred to a paper by C. R. Harrington published in *The British Medical Journal* of December 26, 1936. Harrington showed that the iodine taken up by the thyreoid is introduced into the molecule of tyrosine to form 3:5-diiodotyrosine, and that part of this is further converted into thyroxine, which is "the only naturally occurring pure compound to exhibit the qualitative physiological properties of the thyroid gland". The thyroxine and diiodotyrosine so formed are then linked directly or through other amino-acids and are finally built up into the molecule of thyreoglobulin or colloid (the storage form of thyreoid secretion). The true thyreoid secretion is in all probability a peptide containing both thyroxine and diiodotyrosine, and it is liberated from thyreoglobulin and released into the blood stream in response to the demands of the body. The general effect of the thyreoid secretion is to control the rate of metabolic processes. Harrington pointed out that the functional activity of the thyreoid is possibly subject to an inhibitory control by the suprarenal cortex and that it is undoubtedly influenced by the activity of the anterior pituitary. J. H. Means, in his important book "The Thyroid and its Diseases", shows that the nervous system and the endocrine system have become by the process of evolution the regulating, coordinating, integrating or indeed the governing mechanisms of the higher animal body. We thus have the neurone and the hormone, and "they, together with his inheritance, determine the form, stature and behaviour of man". The thyreoid, therefore, as a member of the group concerned in the coordinating

mechanism cannot logically be considered apart from its endocrine partners. Obviously the partner with which it must be considered is the pituitary—the "leader of the endocrine orchestra"—and this introduces at once the subject of the thyreotropic hormone of the anterior pituitary lobe.

The literature on the thyreotropic hormone is voluminous; readers will remember that Professor C. G. Lambie discussed its many aspects in this journal in December, 1939, in the light of no less than 309 references. Ever since Loeb and Aron, working independently of one another, discovered in 1929 that hyperplasia of the thyreoid gland resembling the histological picture of Graves's disease could be produced in guinea-pigs by injections of anterior pituitary extract a great deal of experimental work has been done on the subject. In this regard it is well to remember the warning, so well given by Means, that we should not lose sight of the fact that since the endocrine system is a product of evolution, its actions will not be identical in any two species. For this reason great caution must always be observed in applying the results of animal experiments to the endocrine physiology of man. In a recent discussion in these pages on the male sex hormones attention was drawn to the peculiar interaction of the body hormones. And here we may recall that if the anterior lobe of the pituitary is removed atrophy of the thyreoid and gonad occurs; on the other hand, if the thyreoid or gonad is removed hyperactivity of the anterior pituitary takes place. Although we are ignorant of the chemical nature of the thyreotropic hormone of the anterior pituitary, we know that it stimulates the thyreoid to intense hyperplasia, purges it of stored colloid and produces the picture of complete thyreotoxicosis. Without the mediation of the thyreoid it has no calorigenic effect, nor can it produce any of the symptoms of thyreotoxicosis. After the thyreoid has responded to thyreotropic hormone for some time, it ceases to be able to do so, or, as Means so well expresses it, "some mechanism comes into play which neutralizes or combats the hormone, shielding the thyreoid from its effect". The upshot of experimental and clinical investigations is that control of the thyreoid function by the anterior pituitary is not complete (Means states that the control is "within limits" and relative rather than absolute); some other factors certainly come into play. The essential characteristic of thyreotoxicosis is overproduction of the thyreoid hormone, and if the thyreotropic hormone of the anterior pituitary had any part in the stimulation of the thyreoid gland to overproduction, it would be expected that some evidence of excess of the anterior pituitary hormone in blood or urine would be forthcoming. As Dr. Byrom has stated, there is no such evidence. As an example of the type of research in this sphere the work of C. L. Cope may be quoted.[1] He investigated the serum and urine concentrates of patients with Graves's disease and failed to find any excess of the thyreotropic hormone; he concluded that the pituitary origin of the disease through a thyreotropic hormone was doubtful. Again, R. W. Ransom and P. Starr,[2] by the investigation of urinary extracts, found slight thyreotropic activity in normal persons, more activity in men whose thyreoids had been removed, but none in persons who were subjects of hyperthyreoidism. They regarded these findings as being opposed to the pituitary thyreotropic origin of Graves's disease. In the absence of direct evidence many will accept the facts as Dr. Byrom has stated them and will go no further. Others may be willing to agree with C. G. Lambie that recent work compels us to recognize the possibility of the existence of varieties of thyreotoxicosis in which the primary seat is outside the thyreoid gland. There is not a great deal of difference between the two views, provided we do not forget and regard a possibility as being comparable with a proven fact. There is an attraction in the building of theories on legitimate grounds as long as we remember that they are theories. It is no doubt for this reason that Means concludes his book with a chapter entitled "Facts and Fancies in Matters Thyroid", a chapter which every medical practitioner could read with profit.

When we leave the world of thyreoid facts to enter that of fancy, a great deal of interest is found to centre around what may be called the thyreotoxic diathesis. Means writes that in matters thyreoid there is not only a hormone with specific actions to be considered, but also a living subject upon which it acts. "The picture presented either in health or disease is conditioned not alone by the properties of the hormone, but by the peculiarities, reactivity, reaction patterns, constitution, and so forth, of the individual in which it works." Sometimes a psychic trauma is followed in such a dramatic fashion by manifestations of thyreotoxicosis that no conclusion other than that of cause and effect can be drawn. Why this should be is a pertinent question and one difficult to answer. In 1929 Warthin wrote of a Graves's constitution. Means not only believes that only certain persons can have exophthalmic goitre, but is willing to declare with Freeman that among those persons who become thyreotoxic the picture will differ from person to person in accordance with the differences in personality. "The intoxication by thyroid hormone, like that by alcohol, the pneumococcus, or what you will, brings out the latent tendencies or suppresses the manifest characteristics of the individual who is intoxicated." Here is scope for discussion and possibly for further flights of fancy. We may ask: "What is personality?" and: "How far, if at all, is it dependent on the endocrine mosaic of the individual?" Again we may ask: "Is the endocrine mosaic inherited and, if it is, can it be altered by agents encountered in a changing environment?" It is reasonable to believe that a response to a certain type of stimulus will vary with the make-up, the attributes, of the body stimulated; it is something like the seed and the soil in a bacterial infection. But if we decide that the vagaries of the endocrine system are responsible for the behaviour of the thyreoid gland in thyreotoxicosis and also go to the extreme length of postulating an influence of the endocrines on the personality, we shall find ourselves in a position of peculiar difficulty and may wonder whether our speculation has been worth while.

The subject of antihormones, in considering which we leave the world of pure fancy for one where a few scattered facts are to be found, has been left to the last because in the opinion of many workers they hold most hope for the future treatment of thyreotoxic conditions. Dr. Byrom summed up the position in one sentence when

[1] *The Quarterly Journal of Medicine*, Volume VII, 1938, page 151, quoted in "The Medical Annual", 1939, page 383.

[2] *Archives of Internal Medicine*, Volume LXI, 1938, page 726, quoted in "The Medical Annual", 1939, page 500.

he said that there were no known antihormones to primary hormones like thyroxine and (as already stated) that although an antihormone to the thyreotropic hormone of the pituitary was known, it could not yet be used in treatment. The chief workers in this field have been Collip and his collaborators at Montreal. They showed that it was possible to produce in animals an immunity to several of the principles of the anterior lobe of the pituitary, including the thyreotropic hormone. Working with the thyreotropic hormone, they found that while at first injections produced a rise in the metabolic rate, repeated injections failed to produce the same effect. Collip showed that when the serum of an animal in which this phenomenon had been noted was injected into another, it would confer on this other animal an immunity to the thyreotropic hormone. Collip's idea is that for every hormone there may be an opposite or antagonistic principle, and that this antagonist is present in the normal subject, but may not be detected until it exceeds in amount the hormone substances with which it is balanced. He and his fellow workers think that the antihormone is a true hormone and has not arisen as a result of a reaction between antigen and antibody. Many investigators have suggested that the opposite view is correct and that the reaction by which the antihormone is produced is really of immunological type. A. T. Cameron, in the fourth edition of "Recent Advances in Endocrinology", states that in most of the experiments such a possibility exists, since protein preparations from animals of one species produce the effect following injection into animals of a different species. Marrian and Butler (quoted in some detail by Cameron), in a review of the subject in 1937, set out the evidence for both views and concluded that it indicated that the antithyreotropic hormone was an antigen. Cameron adds that since Marrian and Butler wrote, the evidence in favour of the antibody view has become stronger. The subject of antihormones really merits a separate discussion, but the few facts that have been presented will serve to show both the complexity of the subject and its importance. Hope for the future, as has already been stated, is commonly held to centre around antihormones. Obviously what is needed is an antihormone to the thyreoid hormone; this hormone is certainly concerned in the production of thyreotoxicosis. Since we are not certain whether the thyreotropic hormone of the anterior pituitary takes any part in the causation of thyreotoxicosis, we are also not certain on what grounds we should expect an antihormone to it to be effective in the treatment of the condition. The real search should not be for an antihormone, but for discovery of the factor (there may be more than one) which first brings about an increase in the thyreoid hormone.

Current Comment.

ACUTE OCCLUSION OF PERIPHERAL ARTERIES.

THE sudden obliteration of the lumen of a large peripheral artery is fortunately not a common occurrence, but when it does happen it is a dramatic episode and one that often endangers the limb and sometimes indeed the life of the patient. G. Saland has recently reported a small series of cases in which treatment consisted of the application to the limb of alternate suction and pressure, a treatment designed to aid blood flow to the endangered limb, and, in Saland's opinion, an exceedingly valuable procedure.[1] The arterial occlusion may be embolic or thrombotic. Those patients who have cardiac disease, but no peripheral arterial degeneration, have almost certainly suffered an embolism. If, however, vascular disease is present and no focus can be discovered from which an embolus may have arisen, then almost certainly the obstruction has been caused by thrombosis. Sometimes, of course, cardiac and arterial disease will both be present, and the nature of the occlusion will be indefinite. From the viewpoint of treatment or the outlook for the limb this uncertainty is, however, of little importance. Saland's series consisted of only 31 patients. The large arteries, such as the femoral, popliteal and external iliac, were involved most often. Less frequently the aortic bifurcation, the anterior tibial, the axillary and the arteries of the hands or toes were the site of the lesion. The onset was marked usually by sudden severe pain in the limb, but sometimes by numbness. The limb distal to the lesion then lost its arterial pulsation, became cold and either pale or grey and cyanosed. Sensation and motion were no longer possible.

The treatment given by Saland is frankly admitted to be the combination of several therapeutic aids previously advocated by other workers. The patient is put to bed and kept warm, and half a grain of papaverine is given intravenously to allay pain and arterial spasm. Alcohol, which aids peripheral vaso-dilatation, is given as one ounce of whisky three times a day. As soon as possible the limb is placed in a suction pressure apparatus so adjusted that during the negative phase the skin becomes pink or the superficial veins fill, while during the positive phase the limb pales or the superficial veins become empty. The apparatus was used if possible for six to twelve hours the first day, with daily reductions to two hours until a total of approximately one hundred hours of treatment had been given. The therapy could not be used if it caused unbearable pain, or if infection, œdema or moist gangrene was present. The results of the treatment are described in some detail. Of the 31 patients, the first eight were not given this treatment, and despite their absurdly small number, are used by Saland as a control series. Saland then summarizes the results in his untreated and treated patients. "The results in untreated cases then give the following figures: A loss of limb in 62·5 per cent of the cases, and a known mortality of 62·5 per cent. In cases conservatively treated, there was loss of limb in only 5 per cent (one case), and a mortality rate of 40 per cent." It is unfortunate indeed that a very interesting report has been marred by the author making reference to percentages in a series of eight patients, a disregard of statistical significance that was strongly deprecated in these columns on July 5, 1941.

Saland's figures are much too small for emphatic conclusions to be drawn. This much can be said of his results: it is promising that a limb was lost by only one in a series of 23 patients, most of whom suffered occlusion of the main artery of the limb. And this can be said of his treatment: it is rational and offers hope where previously little could be entertained. For this reason alone it deserves extensive and thorough trial, and will probably be found to be a measure of real worth. We would emphasize that the earlier it is applied, the better. If the limb can survive the first few days by virtue of its collateral circulation, then this circulation will increase because of the very demand that is made of it. It is during these first days that measures must be adopted to increase blood flow through collateral channels. The method described may possibly achieve this end. We understand that an apparatus apparently similar to that used by Saland is made and sold in Australia, having been designed and used rather for more chronic and less complete peripheral arterial obliteration. It will probably prove worth while in the examples of complete and sudden occlusion which, though not common, are yet not so rare that our hospitals apparently can afford to remain unprepared for them.

[1] *Annals of Internal Medicine*, May, 1941.

Abstracts from Medical Literature.

THERAPEUTICS.

Pneumonia.

C. K. FRIEDBERG (*The Journal of the American Medical Association*, January 25, 1941) describes two cases in which sulphapyridine therapy was used in lobar pneumonia associated with leucopenia. The prognosis in pneumonia with leucopenia of less than 5,000 leucocytes per cubic millimetre is very grave, and even with a leucocytosis of between 5,000 and 10,000 per cubic millimetre the death rate is high. In the author's cases there was little response to serum or other therapy; therefore the use of sulphapyridine held out the only hope of recovery. In the two cases recorded, striking leucopenia was detected, the leucocytes being 700 per cubic millimetre in one case and 2,000 in the other. There had been no response to treatment including massive doses of antipneumococcus serum. Sulphapyridine was given, ten grammes intravenously and seven grammes orally within twelve hours, followed by one gramme orally every three hours in one case. Improvement occurred in twenty-four hours; the leucocytes increased from 700 to 5,900 per cubic millimetre in twenty-four hours and steadily rose to 43,000 within four days. Rapid improvement and recovery followed. The second case followed a similar course, but the dose of sulphapyridine was one gramme every four hours. The leucocytes rose from 10,000 to 13,000 per cubic millimetre, fell again to 7,000, and then varied between 6,000 and 12,000. It is suggested that a leucopenia is not a contraindication to treatment with sulphapyridine.

The Action of Analeptics and Hypnotics.

ERNEST P. PICK (*Journal of the Mount Sinai Hospital*, New York, March-April, 1941) presents a study of the site of action of analeptics and hypnotics in the brain. He summarizes the experiments of many workers and asserts that evidence points to two groups of hypnotics. The first group consists of the barbituric acid derivatives, such as chloretone, "Nirvanol", morphine, scopolamine, ergotoxine *et cetera*, which affect primarily the subcortical brain stem nuclei, particularly those of the thalamic and subthalamic regions, inducing sleep. The second group, comprised mainly of the aliphatic series, such as alcohol, chloral hydrate, chloralose, paraldehyde, amylene hydrate, "Avertin" and bromides, exert their effect primarily upon the sensory cortex and secondarily radiate to the motor cortex. A more exact method for ascertaining the point of action of hypnotics has been found by Feitelberg and Lampl. Delicate thermocouples are introduced aseptically into the brain, brain-stem and internal carotid, and healing is allowed to occur. The temperature differences are measured with a potentiometer and kymograph. Hypnotics inhibit tissue oxidation and reduce the temperature, whereas analeptics increase tissue oxidation. "Cardiazol" increases the temperature of the brain-stem first and then spreads to the cortex. "Cardiazol", therefore, exerts a waking effect primarily in the presence of thalamic hypnotics and secondarily in the presence of cortical hypnotics. Picrotoxin, caffeine, "Metrazol", benzedrine and ephedrine, and "Hexeton" are thalamic analeptics. Camphor and cocaine convulsions originate in the cortex, since convulsions due to them do not occur in decerebrate animals. Experiments indicate that the sleep regulating centre is located somewhere in the grey matter enveloping the third ventricle and the Sylvian aqueduct.

Bee Venom in the Treatment of Chronic Arthritis.

J. L. HOLLANDER (*The American Journal of the Medical Sciences*, June, 1941) has treated forty-two patients suffering from chronic arthritis with an injectable solution of bee venom. In eight cases amelioration of the patient's condition followed. Of the eight patients, two who suffered from rheumatoid arthritis and one who suffered from fibrositis were considerably better. The number of treatments varied from six to thirty-two and extended over periods ranging from four weeks to a year. The length of the treatment and the total dosage showed no apparent correlation with the degree of improvement. Seventeen patients were given injections of foreign protein and were regarded as a control group; three of these manifested lasting improvement. The author draws particular attention to the history of a man who for twenty-three years had suffered from rheumatoid arthritis; his principal occupation for the last eighteen years was keeping bees. He had been stung hundreds of times and had "sold" bee stings to other arthritics, but still had the complaints and deformities characteristic of advanced atrophic arthritis. This patient was given a course of bee venom injections with no improvement at all. He was actually immune to bee stings, there being no local or general reaction following large doses of venom. The author states that the incidence and the degree of improvement in the whole series was very discouraging, and that all but one of the patients, the one suffering from fibrositis, are still seeking relief from their disease. Even though a few patients showed definite and lasting improvement, the author thinks that a painful, tedious, expensive and somewhat toxic form of therapy should yield much better results to justify its continued use.

Barbiturate Intoxication.

J. P. ANDERSON (*Annals of Internal Medicine*, May, 1941) has described the use of picrotoxin in the treatment of twenty patients suffering from barbiturate poisoning. Fifteen recovered. Serious poisoning resulted only when twelve times the usual dose was taken. Coma, excitement, stertor, cyanosis and shallow slow respirations occurred. The stomach was washed out, 10% glucose in saline solution was given intravenously in amounts up to 6,000 cubic centimetres to produce diuresis. In severe cases one cubic centimetre or three milligrammes of picrotoxin were given intravenously each minute until the patient responded to painful stimuli; thereafter it was repeated every hour or every two hours in doses of three to six milligrammes an hour, sufficient being given to keep the corneal reflexes active. Giant bullæ occurred in several cases at points of pressure, within twenty-four hours of the patient's admission to hospital. It was necessary to prevent the patients from lapsing into severe coma, otherwise the chances of recovery were greatly lessened. Pneumonia developed early in two cases. Treatment with sulphapyridine was indicated.

Sulphathiazole and Sulphapyridine.

S. C. WAGONER AND W. F. HUNTING (*The Journal of the American Medical Association*, January 25, 1941) has compared the results obtained in the treatment of pneumonia in infancy and childhood with sulphathiazole and sulphapyridine. One hundred and nine children were treated; 55 children received one and a half to two grains of sulphathiazole per pound of body weight, and 54 received one grain of sulphapyridine per pound. No attempt was made to separate lobar from bronchopneumonia. An attempt was made on the patient's admission to hospital to recover the causative organism by incubation of material from the pharynx. Pneumococci were found in material from 55 patients, hæmolytic streptococci in nine instances, hæmolytic *Staphylococcus aureus* in ten, and non-pathogenic organisms or no culture was obtained in 43. Two deaths occurred among children very seriously ill when treatment commenced. The results obtained so far as fever was concerned were equally good with both drugs. A significant fall of temperature was noted about thirty-six hours after treatment was commenced with both drugs, and clinical recovery occurred on the fifth or sixth day. Clinical observation suggested a better response to sulphathiazole. Three patients had relapses when chemotherapy was discontinued. On the average the drug was given for four or five days. An attempt to maintain a constant level of each drug in the blood was made; this necessitated a daily change in the dose in many cases. An average concentration of four to six milligrammes per hundred cubic centimetres of blood was aimed at and approximated to in this way. Cyanosis was noted in a few cases in both groups; hæmaturia occurred in one of the sulphathiazole groups, leucopenia in two instances. Vomiting occurred in 18 of the sulphathiazole group and 34 of the sulphapyridine group. On two occasions when the patient did not respond to the one drug after forty-eight hours the other drug was substituted with good effect. Complications occurred more often in the sulphapyridine group, but the numbers were not large enough for accurate comparison. The conclusion is that sulphathiazole is as effective as sulphapyridine in the treatment of pneumonia in children.

NEUROLOGY AND PSYCHIATRY.

Civilian Psychiatric Air-Raid Casualties.

THE psychological reaction of civilians to the stress of air raid has been found by Felix Brown (*The Lancet*, May 31, 1941) to fall into certain groups. Normal people may suffer from mild or severe emotional shock, which is best treated by rest and sedatives. In this connexion women are not more affected than men, and acute shock reactions were not found in young children. Of the psychoneuroses due to air raids, the majority were of the hysterical type associated with amnesia for the provoking event. They were generally of the benign type and were successfully treated by psychotherapy or "Evipan"

narcosis. Exacerbations are prone to occur in existing psychoneurotics as the result of air raids. On the other hand, the psychological immunity to air-raid stress in some well-established psychoneurotics has been remarkable. Psychoses following air raids as a rule developed only in persons predisposed to psychotic reactions. The depressive reaction is the commonest of the psychotic states, especially in elderly men. Established psychotics are largely unmoved by air raids; but the author believes that many cases of senile dementia have been precipitated by air raids, especially when old people have had to be removed from their bombed homes. The treatment of the psychoneurotic and psychotic states produced by air raids differs in no way from the treatment administered to similar patients in peace-time.

Idiopathic and Traumatic Narcolepsy.

NARCOLEPSY is a symptom-complex, not a disease, asserts A. Wilson Gill (*The Lancet*, April 12, 1941), who, after briefly summarizing the previous literature, reports four fresh cases. In most cases there is no history of acute illness; but in the absence of cerebral tumour or other gross pathological change, pathological sleep symptoms are often the result of a localized mild encephalitis. Cataplectic symptoms may occur during or after an attack of epidemic encephalitis. Trauma of the skull causing localized damage in the region of the third ventricle or hypothalamus may cause narcolepsy. Prognosis in cases of narcolepsy is uncertain; but the author believes that it is better in the traumatic cases than in those following encephalitis. Treatment is unsatisfactory. The symptoms are uninfluenced by psychotherapy or psychoanalysis. The basic symptoms are organically conditioned, but symptoms produced by suggestion may be superadded or subtracted.

Hypnotic Suggestion.

SANDOR LORAND (*The Journal of Nervous and Mental Disease*, July, 1941) investigates the phenomena of hypnotic suggestion from the psychoanalytical point of view. He concludes that the therapeutic results of hypnotic suggestion are due to a transference situation based on the revival of early childhood dependence of the child and his parents. The emotional state of the therapist is important; and it is important that the therapist should recognize the limitations of the treatment and be aware of all its psychological implications. Patients who are easily hypnotizable exhibit "masochistic yielding" and may become good media. The author believes that a state of superficial hypnosis is preferable for treatment, which should be followed up at the conscious level, working through the social, sexual and emotional conflicts of the patient.

Psychopathological Disorders in the Mother.

THE mother occupies pride of place in the arena of child psychiatry, and her own peculiar psychopathological disorders frequently impede the therapeutic efforts of the psychiatrist. In a group of 488 problem children, Mabel Huschka (*The Journal of Nervous and Mental Disease*, July, 1941) found that 41·6% of the mothers of these children were suffering from psychopathological conditions; and the mother's pathological state was found to be an essential factor in the problems of the child. These mothers, it is claimed, were difficult to treat because they had established the habit of projecting their difficulties on their children. The author pleads for the better understanding of these mother-child relationships, but refrains from attempting to classify the symptomatic behaviour of the mothers investigated in this article.

Epileptics in Air Raids.

J. TYLOR FOX (*The Lancet*, May 17, 1941) has studied the epileptic reactions of a group of 200 adults and 250 children in an institution for epileptics. They were not of low-grade mental defect. Air-raid activity was prominent for several months; aerial activity occurred directly over the hospital, and bombs frequently exploded in the immediate neighbourhood, which occasioned confinement in overcrowded shelters. The scene, as the author claims, was well laid for observation of the effects of unpleasant emotion upon epileptics, and nothing happened. There was no increase in the average number of fits, and there was very little evidence of mental upset. The author concludes that epilepsy is therefore an illness of a physical rather than mental origin.

In a survey of 77 unselected epileptic patients in London over a period of eighteen months' war and six months of heavy air raids, J. Shafar (*ibidem*) found that there was no difference in the incidence of epileptic attacks and that no psychological complications were noted as the result of air-raid stress.

Electrically Induced Convulsions.

R. E. HEMPHILL AND W. GREY WALTER (*The Journal of Mental Science*, April, 1941) report on the treatment of 200 psychotic patients by electrically induced convulsions. The electrical technique is described in detail. Manic-depressive and involutional melancholics made the best response to treatment; but the authors consider that great benefit may be effected in behaviour in chronic institutional cases. Contra-indications and the complications of treatment are discussed. Vertebral fracture occurred but once among the 200 patients treated. The authors suggest that the treatment leads to an improvement of the circulation in psychotic patients. They claim that patients of the retarded type with stagnant circulation showed a greater tendency to improve. They suggest systematic treatment by electrical convulsions for all physically fit patients in mental hospitals who are suffering from non-organic conditions.

Effort Syndrome.

AN intensive study of fifty soldiers, aged from twenty to fifty-four years, has been undertaken by E. Wittkower, T. F. Rodger and A. T. Macbeth Wilson (*The Lancet*, April 26, 1941) in order to assess the relative significance of the various ætiological factors encountered. On the basis of their biographical studies the authors delineated the following five personality types: (*a*) the George Washington-Calvin-Quaker type, characterized by an unusually keen sense of duty and by a rigid superficial and deep morality; (*b*) resigned grousers, with similarly constituted personalities but less repressed in their aggressiveness; (*c*) open rebels, overtly over-aggressive; (*d*) soldiers with inferior physique and obsessional drive; (*e*) hysterical quitters—those who sought escape from an apparently insurmountable situation through dependence. Effort syndrome is primarily an emotional disorder and occurs in persons of an unduly high and severe self-regard. The symptoms result from repressed aggressions and may form the smoke screen of an honourable retreat from the pains of failure in military life. Physical strain, febrile disease and a variety of toxic influences may become interwoven into the ætiological texture. The men in the first two groups have generally been good soldiers and may respond favourably to orthodox psychotherapy. Those of the third group may respond to a limited extent to psychological treatment, which releases their pent-up aggressiveness and diminishes the subconscious conflict involved. The men in the fourth group respond disappointingly, and, being constitutionally handicapped, may have to be discharged from further military service; while the hysterical quitters in the fifth group must be regarded as totally unfitted for military life.

Neurosis, as Viewed by a Regimental Medical Officer.

A. D. LEIGH (*The Lancet*, March 22, 1941), for five months in charge of a large driver-training centre, had the opportunity of observing 400 conscripts every month, 400 volunteers every two months, and a permanent staff of nearly 600 men and members of the Army Transport Service. Fifty-eight cases were psychiatrically studied. Patients with fatigue and exhaustion responded to sedative treatment and returned to duty within a few days. The higher grade mental defectives are difficult to eliminate, but their behaviour abnormalities show up during their training. They are of no value as soldiers. The author believes that there should be no place in the army for men with effort syndrome, chronic stress, dyspepsia or anxiety neurosis. In most cases there is a bad family history or evidence of previous breakdown. The author believes that from the military viewpoint the prognosis is bad, and that there is little hope of recovery while the patient is in the army.

Suprapituitary Adamantinoma and Eunuchism.

Two cases of complete failure of sexual development in patients who reached adult life without exhibiting symptoms in the early stages are reported by Gwenvron M. Griffiths (*Brain*, Volume LXIII, Part 4, 1940). In both cases the third ventricle was distorted by the tumour, but there was no invasion of the ventricular wall. The lesion arose from the pituitary stalk and it involved the supraoptic nucleus. In the male patient, who was aged twenty-four years, the penis was infantile and there was a slight degree of gynecomastia. The endocrine organs were small, especially the testicles. No spermatozoa were found at autopsy. In the case of the female patient, a school teacher of thirty-six years, there was neither breast development nor menstruation. She had had no physical or emotional changes at puberty. There was neither pubic nor axillary hair. The uterus and ovaries were extremely infantile. The author could find no evidence that the control of sexual development is localized in the supraoptic nucleus.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on July 31, 1941, at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. WILFRED VICKERS, the President, in the chair.

Thyreotoxicosis.

DR. S. A. SMITH read a paper entitled "The Treatment of Thyreotoxicosis" (see page 311).

DR. H. R. G. POATE read a paper entitled "The Surgeon's Responsibilities in Thyreotoxicosis" (see page 313).

DR. C. DE MONCHAUX read a paper entitled "Radiotherapy in Thyreotoxicosis" (see page 316).

DR. F. B. BYROM opened the discussion. He said that he had been sorry to hear Dr. de Monchaux plead for the abandonment of the term hyperthyreoidism in favour of the term thyreotoxicosis, because he (Dr. Byrom) intended to make the opposite plea, on the grounds that thyreotoxicosis implied an altered rather than an excessive secretion of thyroxine. There were no known antihormones to primary hormones like thyroxine, and although an antihormone to the thyreotropic hormone of the pituitary was known, it could not as yet be used in treatment. Attempts had been made to detect thyreotropic hormone in the blood and urine of normal and hyperthyreoid subjects, but with inconclusive results, and there was at present no evidence for or against the view that the pituitary was implicated in this disease. Œstrogenic hormone had been used with the aim of damping down pituitary activity, but it was doubtful whether the doses used would have any significant effect. It should also be remembered that the inhibitory effect of œstrogenic hormone on the pituitary was not confined to the thyreotropic activity of the latter. Dr. Byrom went on to say that he had been sceptical about the occurrence of liver damage in thyreotoxicosis, but that in two recent post-mortem examinations on untreated cases of the disease he had observed degenerative changes of a kind which he had not observed in any other condition. He agreed with Dr. de Monchaux that the histological appearances in the thyreoid gave little support to the view that several distinct clinical types of thyreotoxicosis existed.

DR. H. J. DALY referred to anæsthesia in thyreotoxicosis. He said that surgical treatment had formerly been associated with a high mortality rate, but it was now a comparatively safe procedure. This was largely due to the improved preliminary medical therapy and the improvement in surgical technique. In a lesser degree improvements in anæsthesia based on a more complete knowledge of the problems associated with thyreotoxicosis had contributed to better results. Dr. Daly said that he had had very little experience with local anæsthesia, although at the Mayo Clinic it was used extensively. There the anæsthetists carried out the cervical nerve block, and that procedure was time-conserving for the surgeon. The difficulty with local anæsthesia was that it was often inadequate for extensive dissection. Surgery was limited when local anæsthesia was used, and often the patients became restless and troublesome, and general anæsthesia had finally to be used. At the Mayo Clinic general anæsthesia was often used in cases of severe toxicity. At the Lahey Clinic, where formerly local anæsthesia had been used, they had changed to general anæsthesia; they used the closed circuit method, with cyclopropane and oxygen. They also used "Nembutal" or "Omnopon" and scopolamine.

Dr. Daly went on to say that the type of anæsthesia must be chosen with due regard to the physical and psychic disabilities and the biochemical disturbances of the thyreotoxic patient. In most cases it was necessary to employ a judicious combination of methods. The anæsthetic agent and method must be chosen to suit the individual patient. Even if a great improvement in anæsthesia could be claimed, it would be wrong to suggest that a perfect method of anæsthesia had been found. The requirements, risks and difficulties of anæsthesia in the two groups of thyreotoxic patients differed in certain respects. For example, in the primary type of thyreotoxicosis the excitability and nervous instability often overshadowed any circulatory insufficiency, whereas in the secondary type the latter was often the main consideration. In the primary type, therefore, control of the nervous system by some form of preanæsthetic sedation was usually of paramount importance. Basal narcosis was the term applied. The ideal plan was to "steal the thyreoid", and this method should be employed for highly nervous patients. It was often difficult to achieve in public hospitals. The employment of the method resulted in the arrival of patients in the operating theatre with a comparatively slow pulse rate, whereas with the usual methods the pulse was racing. In this type of case it had to be remembered that much larger doses of sedatives were tolerated. When ordinary doses of sedatives were given, these patients were usually awake and restless, and in a state of nervous tension, with a rapid, bounding pulse and a raised temperature. Dr. Daly said that his practice was to give one-third or two-thirds of a grain of "Omnopon" and one one-hundred-and-fiftieth of a grain of scopolamine (lævo-rotatory) one and a half hours before the patient was due in the operating theatre. The patient was made comfortable, lying on his side. Paraldehyde (one drachm per stone of body weight) in three ounces of whey was given *per rectum* one hour before operation. Elevation of the foot of the bed aided retention of the paraldehyde.

Dr. Daly further said that for highly nervous patients (particularly when the gland had to be stolen) "Avertin" was substituted for paraldehyde. The difficulty with paraldehyde was that the patients were not always asleep when they were brought to the operating theatre; but sometimes it was very successful. The dose should not be more than eight fluid drachms, no matter what the patient's weight. Another objection to paraldehyde was the smell; but that did not trouble the patient. Paraldehyde also caused these patients to sweat heavily and become flushed; sometimes it made them thirsty. One advantage of paraldehyde was its low cost. If "Avertin" was used, it was possible to be certain that the patient was asleep. The dose given was in excess of the basal dose, but the patient slept even if he received only the basal dose. "Avertin" cost ninepence per cubic centimetre, and six cubic centimetres were usually required; thus in the average case four shillings and sixpence worth of "Avertin" was used. "Omnopon" (one-third of a grain) or morphine (one-sixth of a grain) was given an hour and a half before the operation, and then a basal dose of 100 milligrammes of "Avertin" per kilogram of body weight was given half an hour before operation. The patient was then moved to the operating table with the least possible disturbance and handling, and local or general anæsthesia was induced. If the patient was deeply asleep, local anæsthesia might prove sufficient, with terminal field block or cervical nerve root block. For the steps of the operation associated with discomfort, a few cubic centimetres of "Pentothal Sodium" were used to subdue the patient. If the patient was restless and not cooperative and responded to the injection of a local anæsthetic agent with undue muscular response, it was usually necessary to resort to a general anæsthetic. At the Prince Henry Hospital and in Dr. Daly's private practice "Avertin" was used for patients with toxic or obstructive goitre or a combination of both. The patients were prepared for operation by the exhibition of bromides and "Luminal" for a week or so beforehand. Dr. Daly gave these patients 10% over the basal dose, 110 milligrammes per kilogram of body weight; morphine was given at the same time, half an hour before the patient was due in the operating theatre. When the patient arrived in the theatre Dr. Daly sprayed the right nostril with a 1% solution of "Decicain", which produced surface anæsthesia. He did not use atropine, because it stimulated the sympathetic, and the sympathetic of these patients was already over-stimulated. After these measures Dr. Daly passed Magill's tube, which he described as one of the greatest advances in the past twenty years. The large rubber tube was passed through the nose and blindly into the trachea, and ensured an airway. No matter what the surgeon did, he could not interfere with the airway, and that was an important consideration in all these operations. It was difficult to hold an air-tight mask, and with the Magill tube these difficulties were at an end. Dr. Daly had never seen a patient stop breathing when this tube was used, and he did not think it caused tracheitis. After the Magill tube was in place the nitrous oxide machine was turned on. Dr. Daly used the closed circuit apparatus, one in which the gases were circulated all the time and the carbon dioxide was absorbed by soda lime. After a minute or two the cyclopropane was turned in, if the patient was in a severely toxic condition, or ether if such was not the case. It was essential that these patients should be kept a good colour. The surgeon could not watch the patient's colour, but the anæsthetist could look at the patient's ears, and he had a much better chance than the surgeon of seeing whether the patient was cyanosed. After the operation a laryngoscope was passed and all mucus and any secretion from the trachea were sucked away, the vocal cords were examined, and the patient did not leave the operating theatre until he coughed. Dr. Daly stressed the point that the surgeon or anæsthetist engaged in performing a thyreoidectomy should be competent to pass an intratracheal tube. The question of laryngeal collapse was serious; it was estimated to occur once in 1,000 cases, and Dr. Daly had had experience of two cases. The obstruction lay beyond the cords, and the only thing to do was to pass a tube

below the obstruction and inflate the lungs with oxygen; or a satisfactory method was to blow through the tube. The lungs were easily distended from the inside.

Dr. Daly then discussed general anæsthesia. He said that it was employed in the great majority of cases; but as Dr. Poate had mentioned, it had to be borne in mind that whatever the general anæsthetic agent used, it should permit of an adequate supply of oxygen to the patient. Patients suffering from either the primary or the secondary type of thyreotoxicosis had to be provided with sufficient oxygen, certainly not less than that present in the atmospheric air. Although nitrous oxide was highly desirable because of the absence of pharmacological effects on the vital organs—for example, the liver, heart *et cetera*—it had to be borne in mind that unless the preliminary sedation had been particularly heavy, its anæsthetic action was so weak that it was impossible to attain any great degree of narcosis without reducing the oxygen percentage so far that anoxia was inevitable. This might be avoided by the use of (i) supplementary local anæsthesia or (ii) cyclopropane or ether, or both, in minimal amounts. With regard to an adequate oxygen supply, Dr. Daly said that Dr. Poate had referred to the use of "Carbogen"; Dr. Daly agreed with him that it was quite unnecessary, and believed that oxygen was the material that was wanted. He referred to the fact that "Carbogen" from some cylinders stimulated breathing with great effect, while that from others did not. "Carbogen" was supposed to consist of 5% oxygen and 95% carbon dioxide, but the percentage of carbon dioxide varied. Dr. Daly thought that there was no way of deciding how much carbon dioxide was in the cylinders. He had discarded "Carbogen" and used oxygen only.

With further reference to cyclopropane, Dr. Daly said that it had certain disadvantages, particularly the tendency to produce excessive bleeding; but its great advantage was that it permitted oxygen percentages three or four times that of the air. The use of ether was restricted to those steps in the operation usually accompanied by reflex disturbances, such as laryngeal spasm and coughing, which were prone to occur during light anæsthesia when, for example, the superior poles were being delivered or when the gland was being rolled medially and dissected from the trachea. Speaking again of general anæsthesia, Dr. Daly said he used the closed circuit gas method with face mask and artificial pharyngeal airway, unless these were otherwise contraindicated by actual obstruction of the airway or by the likelihood of its development during the operation. The advantages of the method were: (i) it permitted a lighter level of anæsthesia than did intubation of the larynx; (ii) it caused less irritation of the larynx and trachea, thereby reducing the incidence of post-operative laryngeal spasm and mucous secretion; (iii) it shortened the duration of the operation; (iv) it permitted inflation of the lungs with high percentages of oxygen. The disadvantages were: (i) the mask might intrude itself in the way of the surgeon; (ii) the method was more tiring and required more skill and attention on the part of the anæsthetist. In some cases of the primary type of thyreotoxicosis and in a number of the secondary type it was possible to manage the anæsthesia safely only by employing the endotracheal technique. The existence of stridor (paralysis of the cord or pressure of a retrosternal tumour on the trachea) or the possibility of compression of the trachea developing during the surgical manipulations was an indication for the use of the endotracheal technique. In certain cases of the primary type, in which the gland was large, hard, rigid and bound down through prolonged administration of iodine, it might be necessary to intubate the larynx, because the manipulations might lead to compression of the trachea. In the secondary type intubation of the larynx with the use of endotracheal anæsthesia was often required, because the adenomatous enlargement of the gland might produce anatomical displacements and deformities in the larynx and trachea. The use of cocaine in the larynx was a valuable step in preventing reflex laryngeal spasm (i) during operation, when the part was lightly anæsthetized, and (ii) when intubation of the larynx had to be carried out for the endotracheal technique. The guiding principle in the anæsthetizing of these patients was the avoidance of anoxia as manifested by cyanosis. Any darkening of the blood called for correction by appropriate measures.

With regard to post-operative treatment, Dr. Daly said that direct laryngoscopy might be needed if general anæsthesia had been used. Oxygen was administered to maintain the patient a bright scarlet colour, and he was not returned to the ward until this was achieved and his breathing was permanently free. Resuscitation therapy was continued on his return to bed, by means of the administration of oxygen and the use of a suitable posture.

DR. A. T. NISBET recalled that in Melbourne in 1936 he had delivered a ten-minute paper on thyreotoxicosis, and afterwards one of the Melbourne surgeons had remarked to him that he understood why Dr. Nisbet saw so many patients suffering from thyreotoxicosis; there were no surgeons in Sydney capable of dealing with them. Dr. Nisbet was interested in the location of areas of endemic thyreotoxicosis referred to by Dr. Poate. When he was in Queensland some years earlier, Dr. Nisbet had worked out that the majority of patients with thyreoid disorders came from the Atherton tablelands, Pittsworth and Lismore, which at that time, medically speaking, drained over the Queensland border. Dr. Nisbet said that he would be interested to hear from Dr. Poate the areas of distribution of endemic goitre in New South Wales, because such knowledge would give some idea of what might be the matter if patients coming from such areas had unexplained and equivocal symptoms.

Dr. Nisbet went on to say that it appeared that the treatment of thyreotoxicosis had developed away from the medical side and become the object of "crude" surgery and irradiation. Radium seemed to have disappeared from the picture; the radium collar was no longer used. The dose was not so easily measured, and the amount of radium necessary to put round the region of the thyreoid gland was of great value and might lead to greater complications than the use of X rays. Dr. Nisbet was sorry to hear Dr. Poate mention 2,000 *r* as the dose for these patients. He said that the technique was varying daily; radiotherapy was a new side of medicine. They were physicians practising radiology. A patient *in extremis* might need a small dose only; the worse the patient's condition, the better he did. To lay down an average dose of 2,000 *r* was not fair, and Dr. Poate had not mentioned the other factors involved in deep X-ray therapy. Dr. Nisbet uttered a plea that that question should be left to the radiologists, who were worried about it and would do their best. Seven years earlier, when a higher voltage deep X-ray therapy apparatus was installed at Sydney Hospital, three deaths occurred after treatment, one in twenty-four hours and one in forty-eight hours. In these two cases no blame could possibly be laid at the door of irradiation. As to the third case, Dr. Nisbet was not sure. He thought it possible that in their enthusiasm the radiologists might have used too large a dose and too high a filtration of copper; too large a dose might have been given in one day, and the dose might possibly have been repeated too often. Whether that had happened or not, they had learned a great deal from the experience and had been very careful ever since. Doses had been reduced to a minimum, and were given at frequent intervals with careful precautions. The Continental and American figures given by various authors showed a greater or less ratio of cures and improvements, as Dr. de Monchaux had mentioned; the results compared favourably with those obtained by surgery. Dr. Nisbet then referred to a clinical meeting that had been held some years earlier, at which Dr. Poate had shown six patients suffering from thyreotoxicosis, who had failed to show much improvement after irradiation; in the next room there were six other patients who had undergone surgical treatment, but had not completely recovered, and had done well with irradiation. Referring to estimation of the basal metabolic rate, Dr. Nisbet said that there was a great deal of discussion at the present time about its value. One could have the estimation made at the Royal Prince Alfred Hospital with a result such as 45%; three days later, if it were made at the Royal North Shore Hospital, the result might be 100%. At the same time, little had been said about X-ray examination of the skull to determine the size of the pituitary fossa before any form of treatment was undertaken. Dr. Nisbet said that he had that day seen a female patient, aged fifty-five years, who had been taking ten grains of thyreoid extract every day for seven years. He asked her whether she would permit him to examine her pituitary fossa, and she agreed. The clinoid process, anterior and posterior, appeared to be somewhat absorbed and the pituitary fossa was enlarged.

DR. RICHARD FLYNN stated that he thought Dr. Smith had been a little ungenerous in his reference to the value of Plummer's contribution to the treatment of patients with thyreotoxicosis, for to him rather than to any other single individual was the present world-wide safety of thyreoid surgery due. Dr. Flynn said that he was disappointed that neither Dr. Smith nor Dr. Poate had referred to patients who were iodine-resistant, a well-recognized condition, and one which on occasions caused real anxiety.

Dr. Flynn stressed the view that iodine therapy was only to be looked upon as pre-operative medication, a fact that was unfortunately still too frequently overlooked. The mortality from thyreoid surgery would be greatly reduced if it could be emphasized that no patient suffering from hyperthyreoidism should receive iodine until he or she had been admitted to hospital and arrangements had been made for surgical treatment.

To Dr. Nisbet, Dr. Flynn said that irradiation and surgical treatment did not balance, because irradiation after surgical

treatment was just the "topping off" of a cure, while surgical treatment after irradiation, on the other hand, frequently gave a much less satisfactory result.

Dr. Flynn then referred to the ligation of the thyreoid arteries as a stage in the surgical treatment of these patients, and said that in his opinion there was still a place for this procedure in the management of bad risk patients.

In regard to set dosage of Lugol's solution in the preparation of these patients for operation, such as five minims three times a day as a routine measure, Dr. Flynn said he felt that there could not be a routine dosage because the patients varied greatly in their physical types and in the degree of severity of their hyperthyreoidism; in the more toxic cases ten minims three times a day would not be too large a dose.

With regard to post-operative use of iodine, Dr. Flynn said that in many cases he thought it was essential. Many times patients who were dangerously ill had been saved by the intravenous administration of Lugol's iodine. Dr. Flynn then referred to a patient with hyperthyreoidism who had had, he thought, malignant exophthalmos. The patient had, when first seen, marked exophthalmos in the right eye and complete ophthalmoplegia in the left eye; a diagnosis of hyperthyreoidism associated with *myasthenia gravis* had been made. The patient reported back recently, much improved after total thyreoidectomy, with the right eye greatly receded and able to move the left eye freely in all directions.

In conclusion Dr. Flynn thanked the speakers for their stimulating papers.

DR. GEORGE BELL also expressed appreciation of the papers read, and said that there were some points that he wished to stress. He thought the point raised by Dr. Poate and Dr. Flynn was important, that patients should be put at rest and given a course of rest before iodine was administered. This was especially important in public hospitals, where there was a call for beds, and iodine was sometimes administered too soon and before the full effect of rest had been obtained. With regard to the ligation of the superior thyreoid arteries, Dr. Bell said that Dr. Bernard Newmarch had used this treatment; but in those days the types of patients attended were frequently those who were almost *in extremis*. For a number of years Dr. Bell gave up the procedure, and then he noticed that Lahey and Dunhill still thought it had a place in thyreoid surgery. He did not know whether it would be practicable to ligate the inferior thyreoid artery; it was not nearly so accessible as the superior artery. With regard to X-ray therapy, Dr. Bell said that he thought it had a place in the treatment of thyreotoxicosis, and he knew that some physicians thought that in mild, early cases it gave good results. He knew of a few cases in which the results had been quite good. Referring to the patients who had died at Sydney Hospital following treatment by powerful X-ray apparatus, mentioned by Dr. Nisbet, Dr. Bell thought that some of these might have been "blasted into eternity". With regard to local anæsthesia, Dr. Bell said that he had been interested in Dr. Daly's remarks on the question of cervical nerve block. Dr. Bell had formerly used cervical nerve block. He now used narco-local anæsthesia, a modification of Kampff's method, which was easier and safer. He wondered what Dunhill did in cases of severe thyreotoxicosis. Dunhill had once said that the use of nitrous oxide and oxygen anæsthesia was easier for patient and surgeon. There was no doubt that the induction of local anæsthesia was a time-consuming method; it was necessary to wait for the local anæsthetic agent to work. He used "Omnopon" for narcosis. Dr. Bell had been interested in Dr. Daly's remarks about paraldehyde; he had himself seen patients come to the operating theatre after the administration of paraldehyde in a condition that could be described only as "fighting drunk". Dr. Bell did not like paraldehyde; if a rectal anæsthetic was used, "Avertin" was probably better. Dr. Poate was to be congratulated on taking the long view of the question of thyreotoxicosis and for referring to the question of prevention. Dr. Bell thought that the subject might well be taken up with the public health authorities. In the goitrous districts there were many cases of nodular goitre of long standing in which thyreotoxicosis supervened. It was essential for all patients, after operation or irradiation, to be kept under observation for a period of at least two years. Dr. Bell always advised his patients to keep in contact with their doctors for two years after treatment. Dr. Bell asked Dr. Smith to give some of the indications for the use of quinidine in the treatment of fibrillation not responding to operation and digitalis, and in conclusion congratulated the speakers again.

DR. W. I. T. HOTTEN added his thanks to the speakers. Referring to Dr. Poate's remarks on anæsthesia, Dr. Hotten said that anæsthesia had played a part, though perhaps one of minor importance, in improving the results in the surgical treatment of thyreotoxicosis. Dr. Daly had largely expressed Dr. Hotten's views, but there were one or two points he proposed to mention. In deciding on the type of anæsthesia to be used in a particular case of thyreotoxicosis, one had to pay due regard to the physical and psychical disabilities. In the primary type the nervous manifestations often predominated, and these patients required adequate preliminary sedation. A combination of "Omnopon" with scopolamine and paraldehyde was sometimes inadequate, and in these cases Dr. Hotten was accustomed to employ a combination of "Omnopon" and "Avertin", perhaps with a 20% increase of the basal dosage of "Avertin". In these cases there was a distinct advantage to be gained from "stealing" the gland. Dr. Hotten had had patients at the Prince Henry Hospital and Royal Prince Alfred Hospital with a resting pulse rate of 120 per minute, and with inadequate premedication they arrived in the operating theatre with a pulse rate up to 160 per minute. A week later, when preparations for the "stealing" of the gland were completed and adequate premedication was employed, they had been brought to the operating theatre with a pulse rate of 120 per minute (their normal resting rate) or even less. Dr. Hotten said that such patients stood sedative drugs in much larger doses than patients of another type; a 25% to 30% increase was needed to quieten them. That statement applied equally to post-operative as well as to pre-operative sedation. With regard to local anæsthesia, Dr. Hotten said that he had opportunities for employing it. He had used cervical nerve block. He found that when the surgeon delivered the upper pole and turned the thyreoid medially, the patient complained. Dr. Hotten then used a weak solution of "Pentothal Sodium" (2·5%), which calmed the patient for a sufficiently long period to allow delivery of the upper pole of the gland. Dr. Hotten said that in regard to general anæsthesia, whatever anæsthetic agent was used, the guiding principle was that the patient should have adequate oxygen. Nitrous oxide was admirable because of the absence of outside pharmacological effects; but it was a very weak anæsthetic agent, and to produce anything like a suitable depth of anæsthesia was impossible without undue reduction of the oxygen percentage. These patients should have at least 20% or more of oxygen. At the Prince Henry Hospital and Royal Prince Alfred Hospital their plan was to give the nitrous oxide with at least 20% of oxygen, and if adequate anæsthesia was not obtained with this amount of oxygen the anæsthetic level was increased with cyclopropane and/or ether. Ether could be added just as the superior pole was being ligated. Dr. Hotten did not agree with the idea of intubating the larynx in every case. He said that that measure prolonged the induction of anæsthesia, increased the disturbance of the larynx, and after the removal of the tube sometimes led to a tendency to laryngeal spasm with associated cyanosis. It also led to undue mucous secretion. In very toxic cases Dr. Hotten used the ordinary face mask of the gas apparatus, and after induction of anæsthesia introduced a pharyngeal airway. With this Dr. Poate did not always agree, but ward sisters assured him (Dr. Hotten) that breathing was much easier after the patient was returned to bed and there was much less mucous secretion when the face mask was used instead of the intratracheal method. In the secondary type of thyreotoxicosis, when respiratory obstruction was present, intubation might be called for fairly often. It might also be necessary in cases in which laryngeal stridor was present, in which the tumour mass was likely to produce obstruction, or in which a retrosternal mass had to be removed. Adequate oxygen was given not only during the operation, but for the subsequent twenty-four hours. Dr. Hotten said that one or two patients had been tided over a very stormy recovery by means of the masks used. (These he demonstrated.) The mask was comfortable and patients tolerated it quite well. Dr. Hotten then referred to post-operative cyanosis. He dealt first with the question of tracheal collapse, mentioned by Dr. Daly. Dr. Hotten agreed with what Cole had written in *Archives of Surgery*, that the occurrence of tracheal collapse in the absence of pathological changes in tracheal rings was almost impossible. Dr. Hotten thought that some of these cases were caused by irritation of the superior laryngeal nerve, which led to some continued spasm of the glottis or to temporary trauma of the inferior laryngeal nerves with abductor paralysis. It was usual to insert a Magill's tube, through which the patient could breathe for twelve hours. If that measure was not successful, the only thing to do was to perform tracheotomy.

DR. H. H. JAMIESON referred to the practice of "stealing" the thyreoid, and said that it had not been sufficiently stressed. To the patient who was nervously very highly keyed up, it was of great help. Dr. Jamieson said that at the Royal North Shore Hospital not one of his patients had known when the operation was to be performed until it was all over, and in no case had it been necessary to

defer the operation. Dr. Jamieson had always thought it wrong for these patients to be in a general hospital ward; he made a point of isolating them, even if they had to be put on verandas and surrounded by screens. Dr. Jamieson went further than Dr. Flynn with regard to the question of giving Lugol's iodine; he thought its administration should not be begun until the surgeon was ready to start his pre-operative treatment.

Dr. Smith thanked those who had contributed to the discussion. He said that he had noticed that the word "crude", applied to the surgical treatment of thyreotoxicosis, had evoked a reaction; but he had not used the word in a derogatory sense. He still thought it the right word to use, in view of ignorance of the cause of thyreotoxicosis. Dr. Smith congratulated Dr. de Monchaux on the well-balanced account he had given of the role of X-radiation in the treatment of thyreotoxicosis. Dr. Smith had noticed that Dr. de Monchaux had said that one of the indications for X-ray treatment was inoperability caused by the state of the patient's heart, and he would like to examine that statement further. If the patient was so ill from the cardiac point of view, it was unlikely that irradiation would do him any good. Indeed, Dr. Smith did not think that any patient was in an inoperable condition from the cardiac point of view unless he was in an extreme state of failure. One of the greatest advances in the treatment of cardiac disease had been in the use of total thyreoidectomy. At the Prince Henry Hospital at present there was a patient, a woman who had been bedridden for some years before thyreoidectomy was performed five years earlier by Sir John McKelvey. She had been suffering from a severe degree of congestive cardiac failure, but since the thyreoidectomy was able to lead a life of moderate activity. Dr. Smith agreed with Dr. de Monchaux on the subject of the post-operative use of X rays. In view of his opinion of the uses of iodine, he held that irradiation could be employed rather than iodine if surgical treatment was insufficient. To Dr. Flynn, Dr. Smith said that he was sorry if he had given the impression that he blamed Plummer for the misuse of iodine. He had intended to say that after Plummer had introduced iodine therapy many people came to the conclusion that this was the definitive and only treatment for thyreotoxicosis. This had led to the indiscriminate use of iodine that had done so much harm. Dr. Flynn had also asked why it was that they did not give an overdose when a patient was acutely ill; he obviously did not agree with one of Dr. Smith's axioms. Dr. Smith did not think that malignant exophthalmos was a manifestation of thyreotoxicosis in the usual sense; that was the type of case he had in mind when he referred to diagnosis being difficult in some patients with ocular signs. He agreed with Dr. Bell and Dr. Flynn that iodine was frequently given too early by the physicians. On that point the proper use of iodine emerged from one of the axioms he had laid down. With regard to quinidine, Dr. Smith said that the thyreotoxic heart possibly provided the instance above all others in which quinidine was particularly valuable. In fibrillation due to thyreoid disease the results obtained by means of quinidine were excellent. Dr. Smith agreed with Dr. Poate that if a trial of digitalis was made for ten to twelve days without favourable results, the time had arrived to use quinidine. Dr. Smith thought there was a certain amount of fear of quinidine that was unjustified. In general, he did not believe that quinidine was likely to be effective in the treatment of fibrillation present for a great length of time; but frequently one was called upon to treat fibrillation due to thyreotoxicosis when it was not of long duration. Often the abnormal rhythm could be changed with quinidine and not by any other means. Quinidine should not be given in the presence of congestive cardiac failure or in long-standing organic heart disease. Apart from these conditions Dr. Smith thought it could be used with complete confidence in all cases of auricular fibrillation due to thyreotoxicosis.

Dr. Poate, in reply, first referred to the question of deep X-ray therapy. He said that Dr. de Monchaux had given an excellent and reasonable exposition of present-day views. Dr. Poate could not understand why the radiologists did not like iodine to be given during the course of treatment. He thought it necessary for these patients to receive sedatives and alkalis and some iodine, because deep X-ray therapy increased the basal metabolic rate during the course of treatment. He considered it useless to have the basal metabolic rate estimated during the X-ray treatment, because it fluctuated so much. It was for that reason that iodine seemed to him necessary to these patients, not in the full dose of three drops three times a day, but perhaps two drops twice a day. Dr. Poate said that he appeared to have trodden on Dr. Nisbet's corns on the question of dosage of deep X-ray therapy; but he had mentioned it purposely. He was one of the first surgeons in Sydney to think that there was virtue in deep X-ray therapy in thyreotoxicosis. He had been anxious to discover what type of deep X-ray therapy and what dosage suited these patients best. He drew Dr. Nisbet's attention to the fact that he had said that the dosage depended on the state of the patient. He had made the offending statement because in the past he had seen a number of patients who had been treated by superficial irradiation with a machine ordinarily used in diagnosis. He had also had patients who had been given small doses at weekly intervals for three doses, and then brought back for another three doses. But he had found over a period of years that the best results followed when large doses were given. Dr. Nisbet had referred to irradiation of the pituitary fossa region. Dr. Poate had had this done in one hundred cases and had been unable to obtain any useful findings, so he gave up this practice. In those unusual and peculiar cases associated with an excess of thyreotropic hormone and hyperpituitarism Dr. Poate thought it was a good idea to have irradiation directed to the pituitary as well as the thyreoid. Dr. Flynn had referred to the iodine-resistant patient. Over a long period of time the only patients of that type Dr. Poate had seen were those who had had too much iodine; they had either taken it themselves or had been given large doses for a lengthy period. It was found that these patients did not respond as usual to further iodine medication, and that was the reason. Dr. Poate had never seen the dramatic recoveries that had been mentioned following the intravenous administration of iodine. The patient who did provide a dramatic reaction following thyreidectomy was the one suffering from acute liver failure. The patients who gave a frightening reaction to thyreoidectomy were those verging on acute liver failure, and in these dramatic results were achieved by the intravenous administration of glucose. With regard to the question of ligation of the superior thyreoid vessels, Dr. Poate thought that there was no virtue in it, especially since the arterial supply of the gland parenchyma was the inferior and not the superior thyreoid. The right thing to do with these people was to keep the fluid balance up and to treat the patient along standard lines as mentioned. Dr. Poate thought that the use of paraldehyde in premedication was good. Dr. Daly and Dr. Hotten had supported his remarks on oxygen requirements. Dr. Poate thought that next in importance to the reintroduction by Plummer of iodine treatment came the appreciation of the oxygen requirements and the use of the closed circuit method of anæsthesia. Dr. Poate stated that this was one of the reasons why he used local anæsthesia only in rare cases such as that of an old man with a failing heart, for whom he had used local anæsthesia very successfully. In certain cases it was preferable to general anæsthesia. In reply to Dr. Hotten and Dr. Jamieson, who had referred to the "stealing" of the thyreoid, Dr. Poate said that it was a trick to be worked occasionally and not as a routine. He found, as time passed and he had more experience, that he had to use the method on fewer and fewer occasions. The patients who were at all apprehensive or who had a tendency to a rapid pulse rate were the ones from whom to "steal" the thyreoid, if fit for operation otherwise. In conclusion, Dr. Poate complimented those who participated in the interesting discussion.

Dr. de Monchaux, in reply, offered his congratulations to Dr. Smith and Dr. Poate on their stimulating papers. He said that there were two further things he might have mentioned, and the first was substernal thyreoid. In Dunedin, New Zealand, there was a goitre clinic every week; goitre was common in the south. Every patient who attended the clinic was radiologically examined, and a percentage were found to have a substernal thyreoid. That implied a third field for irradiation, over the *manubrium sterni*. Secondly, he had not mentioned irradiation of the pituitary region, because some authorities did not agree with it. Dr. de Monchaux thought that this was because many people regarded the pituitary as the conductor of the endocrine orchestra, and it was not good to interfere with the conductor. He had been interested in Dr. Poate's remarks, but did not think that irradiation was always indicated. With regard to Dr. Byrom's remarks concerning thyreoidal hyperplasia becoming convergent rather than divergent, Dr. de Monchaux said that hypertrophy and hyperplasia were found in every form of goitre to some extent; the types were being brought together and correlated. With regard to the administration of iodine *pari passu* with X-ray therapy, Dr. de Monchaux said that he did not like it, because the two forms of treatment had virtually the same effect, involution or regression of the hyperplasia. If the two were given together, it became harder to assess the value of the course of radiotherapy. The basal metabolic rate might indeed rise during the course of X-ray therapy; the inhibitory effect occurred some weeks later. With regard to the question of "stealing" the thyreoid, Dr. de Monchaux wondered whether this came under the heading of petty larceny.

DR. WILFRED VICKERS, from the chair, on behalf of the New South Wales Branch of the British Medical Association, thanked the readers of the papers, which had been instructive and provocative.

Correspondence.

THE RETIREMENT OF DR. OLIVER LATHAM, AND THE MENTAL HOSPITAL LABORATORIES.

SIR: Many generations of medical students in Sydney have passed by a darkish corridor in the basement of the old school. For nearly forty years this has been the approach to two small, rather dim and overstocked laboratories. If you knocked on the door you might hear "Come in" in a high-pitched Irish voice, and you walked in to an Irish welcome, or the door might open and the one and only Fred Hedger would say: "Do you want to see the Doctor? I'll fix it." For these two lived here their life below stairs. Your first visit was a strange experience, for you might suddenly find the subject had changed from gliosis to Kilkenny without a pause. But you went again, and as you shaded your eyes on coming out of the twilight into the glare you realized that here at any rate was enthusiasm, a long neurological experience, tips and tricks and short cuts, and a wealth of illustrative material. And Fred could always find anything, and nothing was too much trouble for either.

And for thirty years they had been there, and before Oliver was the late Froude Flashman for nearly ten years. And now it is finished. And what then?

Let us first see what has been accomplished in these dingy cells. Oliver Latham, of Irish birth, went to Harrow and thence to Sydney, where he graduated in medicine in 1903. He joined the Mental Hospitals Department in 1905, and shortly after went abroad and studied psychiatry and pathology in Scotland, England and Ireland. After his return he joined Flashman, who was the pathologist to the department. Great credit must be given to Anderson Stuart, Jummy Wilson, the Inspector-General and perhaps others for their foresight in founding so far back as the beginning of this century what was really a neurological laboratory. Many will recall with pleasure the urgent and engaging personality of Flashman. In 1910 Latham took charge and began to put out a stream of papers from this laboratory which was continued up to this year, and there are still some in the press. He has studied and described rare or interesting specimens and cases from very varied sources, and has collaborated with many of our leading clinicians and university men. The following bibliography is a credit to the Mental Hospitals Department and to Australian neurology:

"Wright's Method of Section Cutting Applied to the Nervous System", *The Australasian Medical Gazette*, Volume XXVIII, February 20, 1909.

"Quick Methods in the Estimation of Glucose in Urines", *The Australasian Medical Gazette*, Volume XXXV, January 31, 1914.

"A Case of Sudden Dementia from Massive Cerebral Glioma of Unusual Nature", THE MEDICAL JOURNAL OF AUSTRALIA, November 28, 1914; with J. A. L. Wallace.

"A Contribution to the Study of the Ætiology of Disseminated Sclerosis", THE MEDICAL JOURNAL OF AUSTRALIA, September 18, 1915; with J. F. Flashman.

"A Clinical and Anatomical Report of a Case of Friedreich's Disease", THE MEDICAL JOURNAL OF AUSTRALIA, February 17, 1917; with A. W. Campbell and W. F. Lichfield.

"Acute Encephalitis of Unknown Origin", THE MEDICAL JOURNAL OF AUSTRALIA, October 27, 1917; with T. H. R. Mathewson.

"A Case of Neuro-Fibromatosis", THE MEDICAL JOURNAL OF AUSTRALIA, July 31, 1915; with J. Malcolm.

"An Expeditious Bedside Modification of the Widal Test", THE MEDICAL JOURNAL OF AUSTRALIA, August 7, 1915.

"An Inquiry into Some Results Obtained from the Examination of Some 8,000 Mental Patients by the Bordet-Wassermann Test", THE MEDICAL JOURNAL OF AUSTRALIA, May 25, 1918.

"Combined Sclerosis with Heterotopia of the Cord", *The British Medical Journal*, September 27, 1919; with G. E. Rennie.

"The Control of Enteric Fever in Mental Hospitals", *The Lancet*, August 21, 1926; with C. A. Hogg.

"Some Observations on the Examination of the Cerebro-Spinal Fluid", THE MEDICAL JOURNAL OF AUSTRALIA, November 5, 1927 (supplement).

"A Rare Type of Intracranial Tumour", THE MEDICAL JOURNAL OF AUSTRALIA, November 5, 1921; with T. H. R. Mathewson.

"Pernicious Anæmia with Early Spinal Symptoms", THE MEDICAL JOURNAL OF AUSTRALIA, December 23, 1922; with C. B. Blackburn.

"Encephalitis Lethargica", THE MEDICAL JOURNAL OF AUSTRALIA, July 28, 1923; with C. A. Hogg.

"Atavism and Endocrine Peculiarities in Pigs", THE MEDICAL JOURNAL OF AUSTRALIA, August 23, 1924; with J. I. Hunter and G. P. U. Prior.

"Supra-Pituitary Tumour with Fröhlich's Syndrome", THE MEDICAL JOURNAL OF AUSTRALIA, August 23, 1924; with J. I. Hunter and H. H. Nowland.

"A Contribution to the Discussion of the Histological Problems Involved in the Conception of a Somatic and Sympathetic Innervation of Voluntary Muscle", THE MEDICAL JOURNAL OF AUSTRALIA, January 10, 1925; with J. I. Hunter.

"Some Observations on Folin and Wu's Method of Blood Analysis", THE MEDICAL JOURNAL OF AUSTRALIA, June 14, 1924 (supplement).

"Histological Evidence on the Presence of Sympathetic Nerve Endings in Striated Muscle Fibres", THE MEDICAL JOURNAL OF AUSTRALIA, November 26, 1927 (supplement).

"Trophic Glial States in Spinal Cord Lesions", THE MEDICAL JOURNAL OF AUSTRALIA, May 8, 1926.

"Some Thoughts on the Term Acute Disseminated Encephalomyelitis and its Affinities", THE MEDICAL JOURNAL OF AUSTRALIA, November 28, 1931.

"Some Remarks on the Education of Mental Defectives and After-Care Provision for them as well as the Discharged Mental Patient", THE MEDICAL JOURNAL OF AUSTRALIA, October 8, 1927 (supplement).

"Acute Disseminated Sclerosis accompanied by Polio-Encephalitis", THE MEDICAL JOURNAL OF AUSTRALIA, April 30, 1932; with E. W. Fairfax.

"Tumour of the Brain; Spongioblastoma Multiforme", THE MEDICAL JOURNAL OF AUSTRALIA, December 15, 1928; with G. Bell.

"Some Difficulties met with in the Pathological Diagnoses of Encephalitis", THE MEDICAL JOURNAL OF AUSTRALIA, March 22, 1930.

"The Pathology of Two Cases of Sudden Death", THE MEDICAL JOURNAL OF AUSTRALIA, January 22, 1927.

"A Case of Encephalomyelitis", THE MEDICAL JOURNAL OF AUSTRALIA, August 22, 1931; with W. S. Dawson.

The Beattie-Smith Lectures: "Some Activities of a Mental Hospital Laboratory during Thirty Years", THE MEDICAL JOURNAL OF AUSTRALIA, June 9, 16 and 23, 1934.

"Schilder's Disease", THE MEDICAL JOURNAL OF AUSTRALIA, July 27, 1935; with A. W. Holmes à Court.

Report quoted in "Meningo-Encephalitis following German Measles", by R. J. Taylor, THE MEDICAL JOURNAL OF AUSTRALIA, October 9, 1937.

"A Case of Fractured Spine", THE MEDICAL JOURNAL OF AUSTRALIA, July 2, 1938.

"Hæmatomyelia", THE MEDICAL JOURNAL OF AUSTRALIA, April 8, 1939.

"The Role of the Small Intracranial Blood Vessels in the Pathology of Brain Conditions", THE MEDICAL JOURNAL OF AUSTRALIA, February 12, 1938.

"A Self-Inflicted Small Perforating Wound in the Skull causing Ventricular Abscess", THE MEDICAL JOURNAL OF AUSTRALIA, February 26, 1938; with G. E. Phillips and J. McGeorge.

"Some Notes on the Pathology of the Cerebellar System", THE MEDICAL JOURNAL OF AUSTRALIA, February 8, 1941.

"Chronic Parenchymatous Cortical Cerebellar Atrophy", *Brain*, in the press; with B. Hall and K. B. Noad.

"Ependymal Tumour", in the press; with S. Evan Jones.

"The Medulla in Mental Diseases", in the press; with W. S. Dawson.

"The Pathology of G.P.I., with Special Reference to the Action of Diphtheroid Organisms", *Reports of the Laboratory of the Department of Mental Hospitals*, Volume II, Part 1 (with J. F. Flashman); "Heterotopia of the Spinal Cord", *ibidem*, Volume III (with J. F. Flashman); "A Shorter Modification of Wassermann's Serum Test with Criticisms", *ibidem*, Volume III; "Notes on Test Meals in the Insane", *ibidem*, Volume III; "Two Unusual Cases of Cerebral Hæmorrhage", *ibidem*, Volume III; "Some Observations on Cerebro-Spinal Fluids in the Insane", *ibidem*, Volume III; "Cord Showing Primary Degeneration of the Neurone in a Case (Sir A. MacCormick's) with Malignant Cervical Glands", *ibidem*, Volume III; "Hyperplasia of the Interstitial Tissues of the Nervous System in Myasthenia Gravis", *ibidem*, Volume III; "An Enquiry into the General Physical State, including Syphilitic Taint of Some 1,589 Aments in N.S.W. Mental Institutions", *ibidem*, Volume IV; "The Place of Pathology in a Mental Hospital, *ibidem*, Volume V, and THE MEDICAL JOURNAL OF AUSTRALIA, November 3, 1927 (supplement).

And what now? After nearly forty years it is time to make a break. Two dingy laboratories in the medical school may have been a good thing in the first decade of this century, and it proved a flowering plant under Latham; but we should expect bigger and better laboratories now (I know the old tag on this subject), and where and what should they be? The solution is not easy, and any may prove wrong, for a time at any rate, if the right man be not found. But I have not hesitated in my opinion nor altered it in spite of the woeful lack of foresight shown in failing to appoint someone years ago to learn the job from Latham. The Mental Hospital Laboratories should be in the grounds of one of the larger and nearer mental hospitals, as near to as much living and post-mortem material and as many medical officers in the service as is possible. It should be a central laboratory for this State, directly under the Inspector-General and apart from the university, though, of course, with free intercourse with the university departments. It should be equipped for bacteriology, clinical pathology, serology and biochemistry, as well as neuropathology, its main and specialized subject, and it should be the post-graduate laboratory for psychiatrists, so that outlying mental hospitals may have medical officers who can efficiently do the more urgent and less difficult laboratory work. And it should have a good post-mortem department. And a christening as the Flashman-Latham Neurological Laboratories would be a well-deserved tribute to the pioneers.

The future of laboratory work in the mental hospitals is at stake. I have heard that the dispersal of the laboratory is intended, that some of the work may be done in university departments, some by the Board of Health. Possibly the usual false economy will take place. Certainly it will be hard to replace Latham. But we have been saying that for years, and nothing done. Someone may say, and many think, that I know little about mental diseases or hospitals. That is true. But I do know that to tackle a problem one had better go to the spot. And I am wholly disinterested. I pray that more influential men or bodies will take this matter up, before it is too late, with the powers that be, and if so, their motto might well be "Not less expense (for what could have been cheaper), but much more". Who knows but what some great benefit may come to some poor unfortunates? I am not going to indulge in a controversy. If there be critics of my point of view, will someone else take up a pen?

I trust that Oliver Latham will forgive me for this letter. May he live long to enjoy life in his own inimitable way, and still find time to help those who may follow in his footsteps.

Yours, etc.,

143, Macquarie Street, Sydney,
September 5, 1941.

A. H. TEBBUTT.

REFUGEE DOCTORS.

SIR: On September 3 I had this letter to the editor of *The Sydney Morning Herald* on the above subject, as it has been referred to frequently in the lay Press.

So far it has not been published.

Yours, etc.,

Sydney,
September 8, 1941.

T. W. LIPSCOMB.

Refugee Doctors.

Sir: I feel perturbed about reports appearing in the Press that the Government of this State is being urged to amend *The Medical Practitioners Act*, and to permit all and sundry alien doctors to be registered and allowed to practise; apparently the suggestion is on behalf of Central Europeans. We are proud that the founders of the University Medical Schools in Australia set the very highest standards of professional education before granting degrees in medicine, and these standards have always been maintained. I believe that the standard set for Central European qualification is not nearly as rigid as ours, and think it is quite clear that if we allow persons so qualified to practise in this country we shall lower our standard of medical efficiency.

In spite of the large number of Australian doctors who are on active service there is no convincing evidence that adequate medical attention has not been available to the public at large. I am speaking of the domiciliary general practitioner service of which we in Australia are so justly proud. Why then this clamour to admit practitioners of a doubtful standard, and with language disability in addition?

I can well imagine the disgust of our doctors serving their country when they hear that on their return their opportunity to establish themselves in practice would be restricted, because the authorities have allowed these aliens to become established.

Surely no one doubts that these men are aliens in the true sense—refugees they may be from the present Nazi régime, but they are still German nationals, by birth, language and general outlook, and as such anti-British.

Yours, etc.,

September 3, 1941. T. W. Lipscomb.

INFLAMMATORY CONDITIONS OF THE MOUTH AND PHARYNX.

SIR: In the issue of THE MEDICAL JOURNAL OF AUSTRALIA, page 242, of the date August 30, 1941, in the discussion on inflammatory conditions of the mouth and pharynx at a meeting of the Victorian Branch, Dr. Raymond Hennessy stated that "quinsy, as strictly defined, was never seen in patients aged under ten years". Dr. Hennessy gave as his opinion that cases reported as "quinsy" in medical journals as occurring in children about five years of age were intratonsillar and not peritonsillar abscesses.

The details of the following case may be of interest, if only perhaps as an example of "the exception that proves the rule". On March 6 of this year, Dr. A. W. St. Ledger brought a child from the medical out-patient department, whose name was on the waiting list for removal of tonsils and adenoids, to my out-patients at the Mater Children's Hospital, Brisbane. He remarked that had the child been older he would have diagnosed the condition a case of quinsy.

The child was six years of age, had a temperature of 99·8° F., pain on swallowing, and was unable to open the mouth. On examination the appearance was that of a typical quinsy of the left side, the uvula being displaced to the right. Thus two of the conditions postulated by Dr. Hennessy as necessary for the diagnosis of quinsy were present. The swelling was not palpated at this stage, as intense pain was caused by attempting to open the mouth widely enough for this purpose.

The child was given a general anæsthetic by Dr. St. Ledger, a Boyle Davis gag inserted, and the child placed with the head in Rose's position. At this stage the swelling was palpated and found to fluctuate.

The mucosa was then incised in the orthodox site for such cases, the beaks of a pair of St. Clair Thomson's forceps inserted through the incision and opened. About a drachm of thick yellow pus gushed out and was removed by the sucker. By evacuating the abscess completely and using the headlight to examine the cavity, it became obvious beyond any doubt that the abscess was peritonsillar and not intratonsillar in position.

The child was admitted to the ward for observation and its recovery was uneventful.

Yours, etc.,

Inchcolm,
Wickham Terrace,
Brisbane.
September 8, 1941.

HERBERT EARNSHAW.

A NATIONAL MEDICAL SERVICE.

SIR: The medical profession as an organized body should begin to prepare itself immediately for the rapid changes that are coming inevitably upon it. A vague philosophical approach to the subject of change, or a violent oppositional defence of the existing order have their places in debate, but, in all, they are mere expressions of opinion. If we accept the inevitability of approaching change, and are prepared to believe that such change will revolutionize our present system of private practice, let us make a survey, prepare the ground and even, if possible, lay the foundations upon which society can build its new structure of national medicine, in which we can play a willing and active part. The changes which we see dimly in the mist of the future will loom suddenly large and threatening; unprepared we must, in defence, take up a reactionary attitude and strive to maintain that order which we know. In spite of futile and undignified resistance we will have forced upon us a system that will appear to us unjust, because it will undermine our privileged status.

We must be open-minded about the difficulties involved, and make an honest attempt to meet them. A clinical study of the present system of medical practice should involve a survey of the history of its development, an inquiry into the influence of politics, and a comparative study of systems in other countries. We should not hesitate to call in consultation bodies such as trade unions, political parties and other selected associations. These are the sources from

which will spring future changes. It is wiser to accept freely from them that which their idealism in a spirit of cooperation is glad to grant, than be forced to accept that which their vindictiveness in a spirit of exasperation at our stubborn opposition might invent.

It is not to be expected that reform of the present system will be accepted on our terms alone. Our deliberation must not aim at the preservation of our sectional interests, rather must it be directed towards the maintenance of the high standard of efficiency enjoyed by the public today. In that way we can keep the interest and sympathy of the community. It must be always our endeavour to see that any schemes do not bear the stamp of political expediency, and that the care and treatment of the sick, as a State undertaking, do not suffer a Utopian eclipse.

If we pave the way and set up the signposts to the future, we will not be lost again in the legal lanes into whose tortuosities royal commissions delight to lead us.

Yours, etc.,

135, Macquarie Street, Sydney, September 10, 1941. JOHN D. MAUDE.

THE EFFECT OF EXERCISE IN HOT ATMOSPHERES UPON THE SALT-WATER BALANCE OF HUMAN SUBJECTS.

SIR: In estimating capacity for work at high temperatures it is practically only the wet bulb temperature which matters. At 90° wet bulb, work is practically impossible, at 80° or over, very trying; even in New York at times it rises to 83° and, as indicated below, over 75° can be unpleasant. Of course it is influenced by wind, which may make a favourable difference of 2° or 3°.

An interesting sidelight on the tropical Australia probem was furnished some time since by investigations into the work done by miners at high temperatures. It is generally admitted that a wet bulb temperature of over 75° is unpleasant and it is usually assumed that physical work becomes much less efficient at such temperatures. At Broken Hill, New South Wales, miners working at depths of from 300 to 1,100 feet at wet bulb temperature of 75° F. and under, with an average of 70° to 75°, earned 17½ shillings per shift and broke up 3·41 tons of ore per man. At similar depths, but with a wet bulb varying from 75° to 83°, with an average of 77·8°, they earned $15\frac{1}{10}$ shillings and broke up 2·84 tons of ore.

The difference is not as great as might have been expected. The whole of Australia above ground is below 75° F. average wet bulb for a great part of the year and the major part below 75° for the whole year. The figures relating to the Broken Hill mines were obtained from the report of the Royal Commission which investigated the conditions under which the miners worked.

Similar and precise measurements have, so far as I am aware, not been taken above ground in tropical Australia, but I believe the men working on the Townsville wharf showed similar results, which I cannot find.

Yours, etc.,

103-105, Collins Street, Melbourne, C.1, September 10, 1941. JAMES W. BARRETT.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 179, of September 4, 1941.

CITIZEN NAVAL FORCES OF THE COMMONWEALTH.

Royal Australian Naval Reserve.

Appointment.—John Lawrence Bignell is appointed Surgeon Lieutenant, dated 5th August, 1941.

AUSTRALIAN IMPERIAL FORCE.

Australian Army Medical Corps.

Major (Temporary Lieutenant-Colonel) N. H. W. Saxby is appointed to command a Field Ambulance, *vice* Lieutenant-Colonel M. L. D. McKeon, 6th July, 1941.

Captain D. De La F. Henry is seconded, 11th July, 1941.

To be Major.—Captain E. W. Kyle, 11th July, 1941.

Permanent Supernumerary List.

The undermentioned officers are transferred from the Australian Army Medical Corps from the dates shown: Lieutenant-Colonel K. J. G. Wilson, 20th June, 1941, Majors K. S. Richardson, 26th June, 1941, and R. Jeremy and H. M. Maughan, 30th June, 1941, and Captains R. J. Hoy, 20th June, 1941, and E. W. Levings, 25th June, 1941.—(Ex. Min. No. 161—Approved 3rd September, 1941.)

AUSTRALIAN MILITARY FORCES.

AUSTRALIAN ARMY MEDICAL CORPS.

Seventh Military District.

To be Honorary Captain.—Joseph Lionel O'Connor, 5th August, 1941.

Eighth Military District.

Captain (provisionally) J. H. Thorp is transferred (provisionally) from Australian Army Medical Corps, 1st Military District, 10th March, 1941.

Northern Command.

First Military District.

Major H. M. Saxby is appointed an Assistant Director of Medical Services, 25th July, 1941.

Honorary Captain A. E. Mason is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally), 15th July, 1941.

To be Captain (provisionally).—William Charles Day, 4th August, 1941.

Lieutenant-Colonel N. C. Talbot, M.C., E.D., is appointed a Deputy Director of Medical Services and is granted the temporary rank of Colonel, 8th July, 1941.

The following officers are appointed from the Reserve of Officers (A.A.M.C.) on the dates shown and to be Captains (provisionally): Honorary Captains J. H. Thorp, 20th January, 1941; A. V. Henry and G. W. Allan, 2nd August, 1941.

To be Honorary Captain.—Kenneth Boulton Shallard, 23rd January, 1941 (in lieu of the notification respecting this officer which appeared in Executive Minute No. 48/1941, promulgated in *Commonwealth Gazette* No. 50 of 1941).

Eastern Command.

Second Military District.

Captain (Temporary Major) P. L. Jobson is appointed to command a Field Ambulance, 7th July, 1941.

Captain J. T. Anderson is appointed from the Reserve of Officers (A.A.M.C.), 19th September, 1940.

Honorary Captain N271027 T. K. S. Whiting is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally), 17th July, 1941.

Major T. F. Brown, D.S.O., V.D., is appointed from the Retired List, 14th May, 1941.

The dates of appointment of Captain (provisionally) (now Temporary Major) J. I. Robertson and Captain (provisionally) N75884 A. H. Hodge, which appeared in Executive Minute No. 175/1940, promulgated in *Commonwealth Gazette*, No. 190 of 1940, are amended to read "13th April, 1940," and "27th June, 1940," respectively.

To be Lieutenant-Colonel (temporarily).—Major K. C. T. Rawle, 13th July, 1941.

The notification respecting the appointment of William Cecil Rhodes Sim which appeared in Executive Minute No. 11/1941, promulgated in *Commonwealth Gazette* No. 16 of 1941, is cancelled.

To be Honorary Captains.—Adrian Alan Parker Bedkober, 30th July, 1941; and Henry Pearlman, 31st July, 1941.

Honorary Major A. J. Arnott is appointed from the Reserve of Officers (A.A.M.C.), to be Major (provisionally), 6th November, 1940 (in lieu of the notification respecting this officer which appeared in Executive Minute No. 84/1941, promulgated in *Commonwealth Gazette* No. 96 of 1941).

The following officers are appointed from the Reserve of Officers (A.A.M.C.) on the dates shown and to be Captains (provisionally): Honorary Captains N78059 C. M. Ryan, 19th August, 1940, N79653 J. P. E. O'Brien, 2nd December, 1940, S. G. Sandes, G. N. M. Aitkens, P. W. Gill, E. J. T. Giblin, V. R. Harbison and C. W. Coombe, 6th August, 1941.

The provisional appointment of Captain R. Mackey is confirmed.

The date of appointment of Captain (provisionally) N79609 R. D. Puflett which appeared in Executive Minute No. 14/1941, promulgated in *Commonwealth Gazette* No. 20 of 1941, is amended to read "8th October, 1940".

The notification respecting the appointment of Honorary Captain T. W. Miles which appeared in Executive Minute No. 121/1941, promulgated in *Commonwealth Gazette* No. 136 of 1941, is cancelled.

To be Captains (provisionally).—John Hector Willson, 25th August, 1940, and Geoffrey Francis Seymour Davies, 31st July, 1941.

Southern Command.

Third Military District.

The date of appointment of Captain (provisionally) D. E. Gowenlock, which appeared in Executive Minute No. 37/1941, promulgated in *Commonwealth Gazette*, No. 31 of 1941, is amended to read "10th June, 1940".

Majors J. T. Tait, A. F. Maclure, O.B.E., F. D. H. B. Lawton, O.B.E., and M. Gardner are appointed from the Reserve of Officers (A.A.M.C.), 1st August, 1941.

Honorary Majors H. C. Trumble and H. F. Maudsley are appointed from the Reserve of Officers (A.A.M.C.) and to be Majors (provisionally), 1st August, 1941.

Captain C. Friend is appointed from the Reserve of Officers (A.A.M.C.) and to be Major (temporarily), 1st August, 1941.

Honorary Captains E. G. Robertson, C. J. O. Brown, R. F. May and H. F. Praagst are appointed from the Reserve of Officers (A.A.M.C.) and to be Captains (provisionally) and Majors (temporarily), 1st August, 1941.

Honorary Captains L. M. Bryce and W. McL. Smithers are appointed from the Reserve of Officers (A.A.M.C.) and to be Captains (provisionally), 1st August, 1941, and 5th August, 1941, respectively.

To be Majors (temporarily).—Captains (provisionally) T. E. Lowe and E. E. Price, 1st August, 1941.

To be Captain (provisionally) and Major (temporarily).—George Clifford Scantlebury, 1st August, 1941.

To be Captain (provisionally).—Ella Annie Noble Macknight, 1st August, 1941.

To be Honorary Captains.—John Leo King, Albert Ernest Piper, and Edgar Francis John Smith, 1st August, 1941.

The notification respecting the appointment of Captain (provisionally) D. A. Alexander which appeared in Executive Minute No. 141/1941, promulgated in *Commonwealth Gazette* No. 156 of 1941, is cancelled.

Sixth Military District.

Captain (Temporary Major) M. G. Edison is appointed to command a Field Ambulance, 18th March, 1941 (in lieu of the notification respecting this officer which appeared in Executive Minute No. 134/1941, promulgated in *Commonwealth Gazette* No. 149 of 1941).

Western Command.

Fifth Military District.

Honorary Captain A. Siglin is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally), 5th March, 1941 (in lieu of the notification respecting this officer which appeared in Executive Minute No. 78/1941, promulgated in *Commonwealth Gazette* No. 89 of 1941).

To be Captain (provisionally).—Alan Stoller, 10th August, 1941.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

The following Flight Lieutenants are granted the acting rank of Squadron Leader whilst employed in Squadron Leader posts, with effect from dates indicated: L. J. T. Murphy (1st June, 1941), G. S. Colvin, J. C. A. Dent, J. E. Jordan (1st July, 1941).—(Ex. Min. No. 126—Approved 3rd September, 1941.)

Post-Graduate Work.

CLINICO-PATHOLOGICAL CONFERENCE AT SYDNEY.

THE next clinico-pathological conference arranged by the Post-Graduate Directors of Medicine, Surgery and Pathology will be held in the lecture hall at the Prince Henry Hospital, Little Bay, New South Wales, on Monday, September 22, 1941, at 4.30 p.m. The subjects will be cases for diagnosis and discussion. A cordial invitation to be present at the meeting is extended to all medical practitioners.

WEEK-END COURSE IN SURGERY AT SYDNEY.

THE New South Wales Post-Graduate Committee in Medicine will hold a course of instruction in surgery at the Prince Henry Hospital, Little Bay, during the week-end October 25 and 26, 1941. The programme is as follows.

Saturday, October 25.

9.30 a.m. (in Ward 22).—Cases of Interest: The Director of Surgery, Mr. H. R. G. Poate.
11.15 a.m.—"Spinal Anæsthesia in Upper Abdominal Surgery": Dr. W. I. T. Hotten.
12 noon.—"Some Surgical Conditions in the Small Intestine": Mr. E. M. Fisher.
2 p.m.—"Sudden Death": Dr. S. A. Smith.
3 p.m.—"Large Bowel Obstruction": Mr. John S. MacMahon.
4.15 p.m.—"The Cause and Immediate Treatment of Acute Retention of Urine": Mr. R. G. S. Harris.
5 p.m.—"Cæsarean Section": Mr. Alfred J. Gibson.

Sunday, October 26.

9.30 a.m.—"Some Affections of the Hip Joint in Childhood": Sir Robert Wade.
10.15 a.m.—"Tumours of the Breast": Mr. B. T. Edye.
11.15 a.m.—"Consideration of Some of the Lesions in the Vicinity of the Shoulder Joint": Mr. Wilfred Vickers.
12 noon.—"Cholelithiasis (Latent and Active)": Mr. H. C. R. Darling.

The above lectures will be held in the lecture hall, and where the subjects are suitable will be illustrated by lantern slides, demonstrations *et cetera*.

The fee for the course will be two guineas, except in the case of medical officers of the defence forces on full-time service, who are invited to attend the whole or any part of the course free of charge. Application for registration, accompanied by a remittance for the amount of the fee, must be made to the Secretary, New South Wales Post-Graduate Committee in Medicine, the Prince Henry Hospital, Little Bay.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

AT a recent meeting of the Senate of the University of Sydney the following appointments were made: Dr. S. A. M. Tout as an Honorary Demonstrator in Operative Dentistry; Mr. A. J. Tow as Demonstrator in Chemistry; Mr. R. I. Nicholson as Research Chemist in the Department of Medicine.

The Senate has recorded its great appreciation of the continued interest of the McGarvie Smith Trust in the veterinary school of the university. It has thanked the trustees most cordially for a recent gift of £13,500 to provide a new block of buildings for the purposes of the school.

The Union Trustee Company has forwarded to the university an amount of £100, being the legacy bequeathed by the late R. H. Dangar for the benefit of the veterinary section of the university.

The Bank of New South Wales has generously donated the sum of £100 towards the cost of publishing manuscripts relating to poison plants of New South Wales. The research work in this connexion has been carried out under the supervision of the reader in pharmacy, Mr. H. Finnemore.

Messrs. H. B. Gatenby, M.B., B.S., and R. H. Kenny, M.B., B.S., have both qualified for the degree of Master of Surgery (M.S.).

The Senate has approved recommendations from the Professorial Board relating to the constitution of the Faculty of Science. The Faculty of Science will in future include: (*a*) all full-time lecturers, and (*b*) all full-time assistant lecturers of more than three years' standing.

The Senate has approved the procedure outlined by the Faculty of Science for a special course in radio-physics, to be given over a period of six months to undergraduates enlisting in the combatant forces.

The course in radio-physics will be accepted in place of the normal third term in the case of students in the second year in science and engineering, enlisting and accepted for the special course.

Students in the second year in science and engineering who enlist, and are accepted for the first course in radio-physics, commencing about September 15, will, after completing the course satisfactorily, be deemed to have completed second year in science and in engineering.

The Professorial Board has fixed the following conditions for the award of the Agnes Guthrie Prize:

Agnes Guthrie Prize.

Founded in 1941 by a bequest of £1,500 from the late Robert Guthrie to establish a prize at the University of Sydney in memory of Agnes Guthrie.

1. The amount of the prize, which shall be awarded annually, provided suitable candidates present them-

selves, shall be the yearly income from the capital value of the bequest.

2. The prize shall be awarded either—

(a) for medical research work carried out in the preceding two years by a medical graduate of the University of Sydney,

(b) as a grant to assist a medical graduate of the University of Sydney to undertake medical research work.

Other things being equal, it is to be understood that preference will be shown to research work on arteriosclerosis or on allied subjects.

The Permanent Trustee Company of New South Wales, acting as the executor of the estate of the late Mrs. Blanche Dunlop, widow of Dr. Norman John Dunlop, a graduate in medicine of the university, has informed the Senate that under her will Mrs. Dunlop has left property of the value of about £20,000 to the university on trust. The net income from the property is to be used for the payment of university fees of and/or for the support of needy and promising students of British parentage of the Protestant faith in the Faculties of Medicine or Science, or in the study of surgery.

The Senate has decided that the income from the benefaction shall be used to establish bursaries for medical students. The conditions for the award will be submitted by the Vice-Chancellor to the Senate at a future meeting.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Willson, John Hector, M.B., B.S., 1939 (Univ. Sydney), 8, Birriga Road, Bellevue Hill.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Baret, Henri Victor David, M.B., B.S., 1940 (Univ. Sydney), Prince Henry Hospital, Little Bay.

Fox, Walter Raymond Fancourt, M.B., 1939 (Univ. Sydney), 20, First Avenue, Five Dock.

Grace, Stanley St. John Starkey, M.B., B.S., 1939 (Univ. Sydney), 70, Shell Cove Road, Neutral Bay.

Allen, Murree, M.B., B.S., 1933 (Univ. Sydney), 47 Conway Street, Lismore.

Benbow, Thomas Alexander Palmer, M.R.C.S. (England), 1916, L.R.C.P. (London), 1916, 115, Macleay Street, Potts Point.

The undermentioned has applied for election as a member of the South Australian Branch of the British Medical Association:

Hill, John Seymour Thew Tregarther, M.B., B.S., 1941 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.

The undermentioned have been elected members of the South Australian Branch of the British Medical Association:

Bonnin, Lansell, M.B., B.S., 1941 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.

Goodhart, Richard Akhurst, M.B., B.S., 1941 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.

Hendrickson, Douglas Buxton, M.B., B.S., 1941 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.

Jay, Peter Gerald, M.B., B.S., 1941 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.

Jones, Britten Richard Edmond, M.B., B.S., 1941 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.

Lower, Oswald Bertram, M.B., B.S., 1941 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.

Magarey, Ray, M.B., B.S., 1941 (Univ. Adelaide), Royal Adelaide Hospital, Adelaide.

Obituary.

ROBERT JAMES LOOSLI.

WE regret to announce the death of Dr. Robert James Loosli, which occurred on September 9, 1941, at Camberwell, Victoria.

JAMES LEE SELWOOD.

WE regret to announce the death of Dr. James Lee Selwood, which occurred on September 11, 1941, at Brisbane, Queensland.

Corrigendum.

DR. PHILIP A. MAPLESTONE writes from the Calcutta School of Tropical Medicine, Calcutta, in reference to the report of the discussion on his paper in the issue of March 29, 1941. On page 398 Dr. Maplestone is reported, eight lines from the end of the discussion, as having stated that amœbic abscesses were likely to form in the bladder; this obviously should have been the liver. We regret that this error was made.

Diary for the Month.

SEPT. 22.—Federal Council of the B.M.A. in Australia: Meeting at Sydney.
SEPT. 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
SEPT. 24.—Victorian Branch, B.M.A.: Council.
SEPT. 25.—South Australian Branch, B.M.A.: Branch.
SEPT. 25.—New South Wales Branch, B.M.A.: Branch.
SEPT. 26.—Tasmanian Branch, B.M.A.: Council.
SEPT. 26.—Queensland Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

Editorial Notices.

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